SERUM SODIUM AND POTASSIUM IN NEWLY DIAGNOSED ESSENTIAL HYPERTENSIVES

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INTRODUCTION

Hypertension is one of the leading causes of death and disability among adults all over the world. It remains the major risk factor for coronary, cerebral and peripheral vascular disease. Essential hypertension comprises more than 90% of hypertension (1).

Hypertension is an emerging health problem in India. When majority of people come to know that they have hypertension they have already advanced into a stage with target organ damage – a fatal stroke or myocardial infarction or irreversible renal failure. Unfortunately even in developed countries like United States, fifty million people are found to have hypertension. Of these, 70% are aware of their diagnosis, but only 50% are receiving treatment and only 25% are under control (2).

In addition to a primary increase in cardiac function propelled by overactive sympathetic nervous system, primary retention of salt and water by kidney, other factors contributing to hypertension are hereditary predisposition and high sodium and low potassium intake and excretion.

In a country like India, people used to have a diet rich in sodium and poor in potassium. Many studies have shown that a positive correlation exists between serum sodium and blood pressure and a negative correlation exists between serum potassium and blood pressure. They have shown that a decreased intake of sodium and increased potassium intake or both together may be effective in prevention or even treatment of

hypertension. Independent reports on serum sodium and potassium among Indian hypertensive population were lacking and hence the present study was conducted.

REVIEW OF LITERATURE

ESSENTIAL HYPERTENSION:

An elevated arterial pressure is one of the most important public health problems and despite it's widely recognized high prevalence and associated danger, it remains inadequately treated in majority of the patients. It is common, readily detectable, and usually easily treatable and if left untreated can lead to serious morbidity and mortality from cardiac, cerebrovascular, vascular and renal disease. Adequate hypertension control remains elusive because of the asymptomatic nature of the disease for the first 15 – 20 years even as it progressively damages the cardiovascular system (3). Although our understanding of the pathophysiology of hypertension has increased in 90% to 95% of cases, etiology is still mostly unknown (4).

Definition and classification:

Blood pressure is distributed in a typical bell shaped curve within the overall population. As seen in the Multiple Risk Factor Intervention Trial (MRFIT), the long term risks for cardiovascular mortality rise progressively over the entire range of blood pressure, with no threshold that clearly identifies the potential danger. Therefore the definition of hypertension is somewhat arbitrary and usually taken as that level of pressure associated with doubling of long term risks.

According to JNC (Joint National Committee) 7 report, in adults aged18 years and above, systolic blood pressure of <120 mm of Hg and diastolic blood pressure of <80

mm of Hg is normal. Systolic blood pressure of 120 – 139 mm of Hg and diastolic blood pressure of 80 – 89 mm of Hg is prehypertension. In stage I hypertension, the systolic blood pressure is 140 – 159 mm of Hg and diastolic blood pressure is 90 – 99 mm of Hg. In stage II hypertension, systolic blood pressure is ≥ 160 mm of Hg and diastolic blood pressure is ≥ 100 mm of Hg (5).

Table – I

JNC 7 Classification of Blood Pressure for Adults ≥ 18 years*

Sl. No.	**Category**	**Systolic BP (mm of Hg)**		**Diastolic BP (mm of Hg)**
1.	Normal	<120	and	<80
2.	Prehypertension	120-139	or	80-89
3.	Hypertension Stage I	140-159	or	90 -99
	Stage II	≥160	or	≥100

* (Source, JAMA 2003; 289:2560)

Prevalence:

Cardiovascular diseases account for a large proportion of all deaths and disability worldwide. In 1990 there were 5.2 million deaths from cardiovascular diseases in economically developed countries and 9.1 million deaths from the same causes in developing countries. In 1990 in India, out of 9.4 million total deaths, cardiovascular

diseases caused 2.3 million deaths (25%), 1.2 million deaths were due to coronary heart disease and 0.5 million were due to stroke(6). It has been predicted that by 2020, there would be a 111% increase in cardiovascular deaths in India. Hypertension is a major cardiovascular risk factor and important public health problem in the Indian subcontinent and among the South Asians world-wide (7, 8).

Hypertension is directly responsible for 57% of all stroke deaths and 24% of all coronary heart disease deaths. This fact is important because hypertension is a controllable disease and a 2 mm Hg population-wide decrease in blood pressure can prevent 151,000 stroke and 153,000 coronary heart disease deaths in India (9, 10).

The average BP levels in China were systolic: 118±18 mm Hg and diastolic: 76.6±11 mm Hg in a sample of 10,076 urban and rural subjects 35-54 years of age. In India Gupta et al reported mean systolic BP (men: rural 127±14, urban 125±17; women: rural 124±13, urban 126±18) and diastolic BP (men: rural 81±8, urban 81±9; women: rural 80±8, urban 81±12) levels in western Indian urban and rural subjects aged ≥20 years (11). Systolic BP has increased in Indian men aged 40-49 years from 123±11 mm Hg in 1959 to 128.8±17 mm Hg in 1995(12).

Recent studies among Indians have shown a high prevalence of hypertension in both urban and rural areas (13, 14). The prevalence rate of hypertension in India done by various study groups is shown in the Table – II given in the next page.

Table - II

Recent Indian Hypertension Prevalence Studies (BP >140/90) #

First author	Age group	Place	Sample size		Prevalence (%)	
			Men	Women	Men	Women
Gupta R 1995	20-75	Jaipur	1415	797	29.5	33.5
Joseph A 2000	20-89	Trivandrum	76	130	31	41.2
Anand MP 2000	30-60	Mumbai	1521	141	34.1*	-
Mohan V 2000	20-70	Chennai	518	657	14*	-
Gupta R 2002	20-75	Jaipur	550	573	36.4	37.5
Gupta PC 2004	18-60	Mumbai	40067	59522	43.8	44.5

* Gender specific data not available

(Source, J Human Hypertension 2004; 18: 73-78)

Awareness status of hypertension in India is poor. In urban population of Mumbai there was a very low awareness of hypertension and only 6.1% males and 10.1% females were aware of the hypertension in early 1990`s (15). In Jaipur also it has been reported that only 11% of male and 16% of female hypertensives were aware of their condition (16). Higher awareness of hypertension has been reported among the more educated populations in Kerala and among Parsis in Mumbai (17, 18). In community dwelling elderly individuals in Kerala (n= 357, mean age 70 years) hypertension was present in 51.8% subjects and 44.9% of these individuals were aware of their condition (17). Bharucha and Kuruvilla (18) reported that 53% of men and 44% of women were unaware of their hypertensive status although 905 had their BP measurement in their past. This level of awareness is similar to reported from many developed countries and shows that within India there is a wide variation in hypertension awareness status.

Natural History and Complications:

The pathological hallmark of untreated hypertension is acceleration of atherosclerosis. The higher the BP, the more likely that various cardiovascular diseases will develop prematurely. If untreated, 50% of the hypertensive patients die of coronary artery disease or congestive cardiac failure, about 33% of stroke and 10 – 15% of renal failure. A meta-analysis of nine major prospective studies shows a direct continuous and apparently independent association of diastolic BP with both coronary artery disease and stroke (19).

In general the vascular complications of hypertension can be considered as either hypertensive or atherosclerotic.

I. Hypertensive Complications:

1. Accelerated malignant phase
2. Hemorrhagic stroke
3. Congestive heart failure
4. Nephrosclerosis
5. Aortic dissection

II. Atherosclerotic Complications:

1. Coronary artery disease
2. Sudden death
3. Arrhythmias
4. Atherothrombotic Stroke
5. Peripheral vascular stroke

Overall Cardiovascular Risk:

The degree of risk from hypertension can be categorized with reasonable accuracy by taking into account

1. The level of Blood Pressure.
2. The biological nature of hypertension based on target organ damage.
3. The co-existence of other cardiovascular risk factors (20).

The goal of anti-hypertensive therapy should not only be the reduction of blood pressure but also treating other risk factors. The major cardiovascular risk factors indicated in JNC-7 report are

1. Hypertension
2. Cigarette smoking
3. Obesity
4. Physical inactivity
5. Dyslipidemia
6. Diabetes mellitus
7. Microalbuminuria or estimated GFR <60 ml/min
8. Age (>55 for men, >65 for women).
9. Family history of premature cardiovascular disease (<55 for men, <65 for women)

Systolic Hypertension and Pulse Pressure:

Systolic blood pressure rises in a linear fashion with age, where as diastolic blood pressure increases until the age of fifty then levels off and even begins to fall. Isolated diastolic hypertension is more common in younger subjects, while isolated systolic hypertension emerges as the most common form of hypertension in the elderly. The underlying pathological process is loss of arterial elastic tissue, which means that the pressure was created by left ventricular contraction, can no longer be damped by the aorta and major vessels. Systolic blood pressure is a better predictor of cardiovascular risk and

isolated systolic hypertension is now recognized to be an independent risk factor of cardiovascular disease. A wide pulse pressure has a similar influence on prognosis (21).

Gender Differences:

Hypertension is an important risk factor for cardiovascular disease in women. Although premenopausal women have lower blood pressure than age matched men, the prevalence of hypertension is higher in women than men after the age of sixty five. Obesity is significantly more common in middle aged and older women and is likely to contribute to cross over in prevalence. Oral contraceptive pills increase the risk of hypertension in younger women. Hormone replacement therapy does not raise the blood pressure in women who are normotensive at the start of treatment.

The ratio of hypertension frequency in women versus men increases from 0.6 to 0.7 at age 30 to 1.1 to 1.2 at age 65 years.

Hypertension in black populations:

Hypertension is most common in black than in white and more common in urban than rural blacks. Black individuals have a higher incidence of salt sensitive hypertension than white individuals and retain more sodium leading to expanded plasma volumes and lower plasma renin activity. The complications of hypertension also tend to be different in blacks with a higher incidence of left ventricular hypertrophy, stroke, renal failure and lower risk of coronary artery disease. The increased frequency of left ventricular

hypertrophy and stroke and renal failure is due to severity of hypertension in black and lower risk of coronary artery disease than white is due to more favourable lipid profiles.

Mechanisms of Primary Hypertension:

No single or specific cause is known for most cases of hypertension, and the condition is referred to as primary in preference to essential. Blood pressure is the product of cardiac output and peripheral vascular resistance (BP = CO X PVR). Since persistent hypertension can develop only in response to an increase in cardiac output or a rise in peripheral resistance, defects may be present in one or more of the multiple factors that affect these two forces. This can be described under the following headings.

1. Non renal factors
2. Renal factors

I. Non renal factors:

Primary hypertension is a complex multifactorial and polygenic disorder that results from an interaction between an individual's genetic background and various environmental factors.

1. Genetic Predisposition:

In studies of twins and family members in which the degree of familial aggregation of blood pressure level is compared with the closeness of genetic sharing, the genetic contributions have been estimated to range from 30% to 60% (22). Harrap

suggested that “the average population blood pressure is determined by the environment but the blood pressure rank within the distribution is decided largely by genes (23).

Epidemiological data suggest that for population variability in blood pressure genetic factors contribute 30 – 35%, common household environment about 10 – 15% and non familial factors for the remaining 50 – 55% (24).

If genetic markers of a predisposition for the development of hypertension are found, then specific environmental manipulations could then be directed toward the susceptible subjects (25). Pratt, from his observation of bimodal distribution of blood pressure in some families with hypertensive subjects, proposed autosomal dominant mode of inheritance. Pickering proposed that blood pressure is a quantitative trait with genetic contribution which is polygenic.

Genome wide scanning strategy in sib-pairs has identified chromosomal regions on chromosomes 6, 5, 12 and 15 which showed significant linkage to genes that influences inter individual blood pressure variation. There are several candidate genes within the identified group (26).

Polymorphism of genes involving the RAS system, aldosterone synthesis and adrenergic receptors has been noted to be more common in the hypertensive than normotensive patients (27). Genetic abnormalities may be monogenic in some rare forms

of hypertension like glucocorticoid remediable aldosteronism, Liddle syndrome, and apparent mineralocoricoid excess (28).

2. Fetal environment:

Low birth weight as a consequence of fetal under nutrition is followed by an increased incidence of high blood pressure later in life with an overall estimate that a 1 kg lower birth weight is associated with a 2 to 4 mm Hg higher systolic blood pressure in adulthood (29). Brenner and Chertow hypothesized that a decreased number of nephrons from the intrauterine growth retardation could very well serve as a permanent irreparable defect that eventuates in hypertension (30).

3. Vascular Remodeling:

A number of factors increase peripheral resistance by both functional contraction and vascular remodeling and hypertrophy. Multiple vasoactive substances act as pressure-growth promoters resulting in both vascular contraction and hypertrophy simultaneously, but perpetuation of hypertension involves hypertrophy. Lever and Harrap (31) postulated that primary hypertension has two mechanisms similar to secondary hypertension (a) a growth promoting process in children, and (b) a self-perpetuating mechanism in adults.

4. Neurohumoral causes of primary hypertension:

A large number of circulatory hormones and locally acting substances may be involved in the development of hypertension which causes hypertension by vascular hypertrophy, capillary rarefaction and impaired microvascular dilation (32).

A. Sympathetic Nervous Hyperactivity:

Young hypertensives tend to have increased levels of circulating catecholamines, augmented sympathetic nerve traffic in muscles, faster heart rate and heightened vascular reactivity to α adrenergic agonists (33).

These changes could raise blood pressure in a number of ways – either alone or in concert with stimulation of renin release by catecholamines or by causing arteriolar and venous constriction or by increasing cardiac output or by altering the normal renal pressure – volume relationship.

B. Renin – Angiotensin System:

Both as a direct pressor and as a growth promoter, the renin – angiotensin mechanism may be also involved in the pathogenesis of hypertension. All functions of renin are mediated through the synthesis of angiotensin II. This system is the primary stimulus for the secretion of aldosterone and hence mediates minerlocorticoid responses to varying sodium intake and volume overload. When sodium intake is reduced or effective plasma volume shrinks the increase in renin – angiotensin II stimulates

aldosterone secretion, which in turn is responsible for a portion of the enhanced renal retention of sodium and water.

When large populations of hypertensives are surveyed, only about 30 percent have low plasma renin activity levels, where as 50 percent have normal levels and the remaining 20 percent have high levels (34).

Normal and high renin hypertension:

Some persons with primary hypertension have normal or high renin levels. The concept of "nephron heterogeneity" (35) descried by Sealy and colleagues, assumes a mixture of normal and ischemic nephrons caused by afferent arteriolar narrowing. Excess renin from the ischemic nephrons could raise the total blood renin level to varying degrees and cause high renin levels in patients with primary hypertension.

C. Hyper insulinemia / Insulin resistance:

An association between hypertension and hyperinsulinemia has been recognized for many years, particularly with accompanying obesity but also in about 20 percent of non obese hypertensive patients (36). The hyperinsulinemia of hypertension arises as a consequence of resistance to the effects of insulin on peripheral glucose utilization. This association does not apply to pima Indians but it has been found in blacks, Asians and as well as whites. The impairment of the peripheral actions of the insulin result from a defect in the usual vasodilatory effect of insulin mediated through increased synthesis of nitric oxide, which normally counters the multiple pressor effects of insulin (37). These

pressor effects include activation of sympathetic activity, a trophic action on vascular hypertrophy, and increased renal sodium reabsorption.

The failure of vasodilation to antagonize the multiple pressor effects of insulin presumably eventuates in a rise in blood pressure that may be either a primary cause of hypertension or, at least, a secondary potentiator.

D. Endothelial Cell dysfunction:

The endothelium is now known to be the source of multiple relaxing and contracting substances, of which nitric oxide is an important vasodilator (38). The impairment of normal vasodilation in the insulin resistance syndrome has been shown to involve failure to synthesize the normal endothelium derived relaxing factor (NO).

Nitric Oxide:

Hypertensive patients have been shown to have a reduced vasodilatory response to various stimuli of nitric oxide release that appears to be independent of the etiology of the hypertension and the degree of the gross vascular structural alteration. Impaired nitric oxide mediated vasodilation may promote abnormal vascular remodeling and may be involved in the greater propensity for vascular damage in blacks than in whites. Nitric oxide – mediated forearm responsiveness has been restored by normalization of blood pressure by anti hypertensive drugs with different modes of action.

Endothelin:

Endothelin – 1 causes pronounced and prolonged vasoconstriction and because blockade of its receptors improves endothelium – dependent vasodilation in hypertensive patients (39).

E. Minerals:

Excess of lead and changing ratios among dietary sodium, potassium, calcium and magnesium have also been postulated in the pathogenesis of primary hypertension (40).

II. Renal retention of excess dietary sodium:

A considerable amount of circumstantial evidence supports a role for sodium in the genesis of hypertension. To induce hypertension, some of the excess sodium must be retained by the kidneys. Such retention could arise in a number of ways.

1. Nephron heterogeneity, described as the presence of a sub population of nephrons that is ischemic either from afferent arteriolar vasoconstriction or from an intrinsic narrowing of the lumen. Renin secretion from this sub group of nephrons is tonically elevated. This increased renin secretion then interferes with the compensatory capacity of intermingled normal nephrons to adaptively excrete sodium and consequently, over all blood pressure homeostasis (35).

2. A decrease in the filtration surface by a congenital or acquired deficiency in nephron number or function.

3. A resetting of the normal pressure – natriuresis relationship – the Guyton hypothesis (41).

4. An acquired inhibitor of the sodium pump or other abnormalities in sodium transport (42).

5. Defensive responsiveness to atrial natriuretic hormones (43).

Association of hypertension with other conditions:

1. **Physical inactivity:**

Physical fitness may help prevent hypertension and persons who are already hypertensive may lower their blood pressure by means of regular isotonic exercise. The relation ship may involve insulin resistance because an increased resistance was coupled with low physical fitness in normotensive men with a family history of hypertension.

2. **Alcohol:**

Alcohol in larger amounts (more than two portions a day and more so when drunk in binges) increases blood pressure and arterial stiffness. The pressure effect of larger amount of alcohol reflects an increase in cardiac output and heart rate, possibly a consequence of increased sympathetic nerve activity. Alcohol also alters cell membranes and allows more calcium to enter, perhaps by inhibition of sodium transport.

Alcohol in small amounts (less than one or two usual portions a day) provides protection from coronary disease, congestive heart failure, stroke, and dementia (44). And at least in women, reduces the incidence of hypertension (45). The reduction in coronary disease in persons who ingest small amounts of alcohol may reflect an improvement in lipid profile, a reduction in factors that encourage thrombosis, and an improvement in insulin sensitivity.

Framingham study showed small overall correlation between alcohol intake and blood pressure, but prevalence of hypertension was about two times higher among persons drinking sixty ounce or more of ethyl alcohol per month than among those drinking less than thirty ounce per month (46).

3. **Smoking:**

Cigarette smoking raises blood pressure, probably through the nicotine induced release of norepinephrine from adrenergic nerve endings. In addition, smoking causes an acute and marked reduction in radial artery compliance independent of the increase in blood pressure. When smokers quit, a rise in the blood pressure may occur, probably reflecting a gain in weight. Numerous studies have shown that smokers are thinner than non smokers and that smoking reduces weight. However they will have larger waist hip ratio than non smokers (47).

4. **Hematological Findings:**

Higher hematocrits are found in hypertensive persons and are associated with abnormal left ventricular filling on echocardiography (48). Whole blood viscosity in increased by about 10 percent in persons with untreated mild hypertension, comparable to the increase in their peripheral resistance (49). Pseudo or stress polycythemia with high hematocrit and increased blood viscosity but contracted plasma volume, as well as normal red cell mass and serum erythropoietin levels are found in hypertension. High WBC count is predictive of the hypertension (50).

5. **Hyperuricemia:**

Hyperuricemia is present in 25 percent of individuals with untreated hypertension, more than 75% of patients with malignant hypertension which are about five times the frequency found in normotensive persons (51). Hyperuricemia probably reflects decreased renal blood flow, presumably a reflection of nephrosclerosis.

6. **Sleep Apnea:**

Snoring and sleep apnea are often associated with hypertension, which may in turn be induced by the increased sympathetic activity and endothelin release in response to hypoxemia during apnea (52).

7. **Hypercholesterolemia:**

Hypercholesterolemia frequently coexists with hypertension, at least in part because it impairs endothelium – dependent vasodialtion. Lipid lowering therapy restores

the bioavailability of nitric oxide, reduces arterial stiffness, and lowers blood pressure (53).

8. Obesity:

The majority of patients with high blood pressure are overweight and hypertension is about six times more common than it is in lean subjects (54).

A 10 kg higher body weight is associated with a 3 mm Hg higher systolic and 2.3 mm Hg higher diastolic blood pressure. These increases translate into an estimated 12% increase in the risk of coronary artery disease and 2.24% increase in the risk of stroke.

Body mass index in widely used as a correlation with excess body fat or adiposity but it does not convey information on required fat distribution. Body fat distribution plays a role as a risk factor for hypertension. An increase in waist hip ratio (WHR) > 0.95 in male and 0.8 in female is an independent risk factor for the development of hypertension and is independently associated with hypertriglyceride and increased apoprotein – B. The waist circumference may be the better indicator of visceral fat than waist hip ratio.

Hip circumference – It is measured at the point of one third of the distance between the anterior superior iliac spine and the patella.

Waist Circumference – It is measured half way between superior iliac crest and rib cage in mid axillary line. It correlates well with the systolic blood pressure and diabetes mellitus.

Body mass index = weight in kg / height in m^2.

Table - III

Proposed classification of weight by BMI in adult Asians (55) *

Classification	**BMI (kg / m^2)**
Underweight	<18.5
Normal range	18.5 – 22.9
Over weight	23
At risk	23 – 24.9
Obese I	25 – 29
Obese II	>30

* (Source, Am J Clin Nutr. 2000; 72: 1067-1068)

Metabolic syndrome:

According to ATP III, metabolic syndrome is defined as a cluster of cardiovascular risks in a silver individual – normally hypertension, diabetes mellitus, abdominal obesity, low HDL, increased TGL, procoagulant tendency and increased small LDL.

Obese individuals with high waist hip ratio have high incidence of hypertension than do with low waist hip ratio (56). Visceral obesity is a strong risk factor for hypertension. Although body mass index is a very strong determinant of blood pressure, a visceral distribution of fat has an even greater relationship with the development of hypertension (57).

Sodium Metabolism:

The major extra cellular fluid particles are sodium and its accompanying anions chloride and bicarbonate, whereas potassium and organic phosphate esters (adenosine triphosphate, creatine phosphate, and phospholipids) are the predominant intracellular fluid osmoles. Since sodium is largely restricted to the extra cellular compartment, total body sodium content is a reflection of the extra cellular fluid volume.

Sodium Balance:

Sodium is actively pumped out of cells by the Na + k+ ATPase pump. As a result, 85 to 90% of all sodium is extra cellular, and the extra cellular fluid volume is a reflection of total body sodium content. Changes in sodium concentration generally reflect disturbed water homeostasis, whereas alterations in sodium content are manifest as extra cellular fluid volume contraction or expansion and imply abnormal sodium balance.

Sodium Intake:

Individuals eating a typical western diet consume approximately 150 mmol of sodium chloride daily. This normally exceeds basal requirements. Therefore dietary

intake of sodium results in extra cellular fluid volume expansion, which in turn promotes enhanced renal sodium excretion to maintain steady state sodium balance.

Sodium Excretion:

The regulation of sodium excretion is multifactorial and is the major determinant of sodium balance. Tubule sodium reabsorption is the major regulatory mechanism controlling sodium excretion. Almost two-thirds of filtered sodium is reabsorbed in the proximal convoluted tubule. Further reabsorption (25 to 30%) occurs in the thick ascending limb of the loop of Henle via the apical Na+-K+-$2Cl^-$ co transporter. Distal convoluted tubule reabsorption of sodium (5%) is mediated by the thiazide-sensitive Na+-Cl^- cotransporter. Final sodium reabsorption occurs in the cortical and medullary collecting ducts; the amount excreted being reasonably equivalent to the amount ingested per day.

Sodium and Hypertension:

1. **Sympathetic Nervous System and Renal Sodium Handling:**

The renal sympathetic nervous system directly stimulates sodium reabsorption and renin release from the juxtaglomerular apparatus. Several studies have linked sympathetic nervous system hyperactivity with greater than normal increases in blood pressure in response to a given sodium load (58). Most authorities believe that the mechanism by which the kidney causes hypertension is impairment in the excretion of sodium (59, 60). This impairment may be related to genetic changes in various sodium exchangers in the proximal and distal tubules that result in altered responses to

stimulation by the sympathetic nervous system and the renin angiotensin aldosterone system.

Interventional studies with sodium restriction and / or loading have revealed that the blood pressure responses in many hypertensive patients are salt sensitive. Salt loading of patients with essential hypertension results in an increase in blood pressure and in a net total body sodium accumulation.

2. An increase in cytosolic free sodium concentration in cells of hypertensive patients compared with age and sex matched normotensive controls have been documented (61). This results from altered activity of the Na+ / H+ antiporter and the Na + / Li+ counter transporter. This increase in intra cellular sodium is highly correlated with the presence of an elevated diastolic blood pressure.

3. Most patients with essential hypertension have a defect in the pressure natriuresis curve, in which higher systemic pressures are required to excrete a sodium load (62).

4. Another mechanism for decreased sodium excretion in patients with essential hypertension is an enhancement of tubulo glomerular feed back (63).

5. Alterations in intrarenal vasoactive mediators may be involved in the impairment of sodium excretion in patients with essential hypertension. There may be low levels of renal vasodilators, such as prostaglandins, dopamine, and nitric oxide as well as elevated

levels of renal vasoconstrictors such as angiotensin II and adenosine and increased activity of the renal sympathetic nervous system. Alterations in the levels of these agents could contribute to net sodium reabsorption because of their direct effects on tubular sodium transport.

Potassium Balance:

Potassium is the major intracellular cation. The normal plasma potassium concentration is 3.5 to 5 mmol / L, whereas that inside the cells is about 150 mmol / L. therefore, the amount of potassium in the extra cellular fluid is less than 2% of the total body potassium content. This is due to the principal result of the resting membrane potential and is crucial for normal neuromuscular function. The basolateral Na+ K+ - ATPase pump actively transports potassium in and sodium out of the cell in a 2:3 ratio, and the passive outward diffusion of potassium is quantitatively the most important factor that generates the resting membrane potential.

Potassium Intake:

The intake of individuals on an average western diet is 40 to 120 mmol/d 90% of which is absorbed by the gastrointestinal tract. Immediately following a meal, most of the absorbed potassium enters cells as a result of the initial elevation in the plasma potassium concentration and facilitated by the insulin release and basal catecholamine levels.

Potassium Excretion:

Renal excretion is the major route of elimination of dietary and other sources of excess potassium. 90% of the filtered potassium is reabsorbed by the proximal convoluted tubule and the loop of Henle. Proximally, potassium is reabsorbed passively with sodium and water, whereas the luminal Na+ - K+ - 2Cl – co transporter mediates potassium uptake in the thick ascending limb of the loop of Henle. Therefore potassium delivery to the distal nephron approximates dietary intake. The cell responsible for potassium secretion in the late distal convoluted tubule and cortical collecting duct is the principal cell.

Modern life style contributes to excess sodium intake. Since sodium excess is associated with fluid restriction and increased blood volume which in turn results in an increase in blood pressure especially in salt sensitive individuals. At the same time modern living with refined food leads to reduced potassium consumption. Hence there is a need to study sodium and potassium levels among newly detected essential hypertension from different parts of India to find out the true scenario and the status. The present report provides an insight into one of its kind from South India.

AIMS & OBJECTIVES

1. To study the levels of sodium and potassium in patients with primary hypertension.

2. To correlate the sodium and potassium levels with blood pressure.

MATERIALS AND METHODS

Setting : The work was carried out in the outpatient department and medical wards of Govt. Rajaji Hospital, Madurai.

Design of the study : Analytical study

Period of the Study : One year – February 2005 to January 2006

Sample size : 100 cases (70 cases and 30 controls)

Ethical committee approval : The present project was approved by the ethical committee.

Inclusion criteria:

1. Patients with primary hypertension.
2. Patients whose age was above 18 years were included.
3. Both sexes were included.

Exclusion Criteria:

1. Patients below 18 years.
2. Patients with renal failure.
3. Pregnancy.
4. Patients with secondary hypertension.
5. Patients on non-steroidal anti-inflammatory agents, antihypertensives, diuretics, beta blockers or stimulants.
6. Patients with malignant hypertension.
7. Females on oral contraceptive medication.
8. Patients with peripheral vascular disease.

9. Patients with diabetes mellitus.
10. Patients with acute diarrhoeal diseases.

Consent:

The study group thus identified by the above criteria (inclusion and exclusion criteria) was first instructed about the nature of the study. Willing participants were taken up after getting a written informed consent from them.

Study Subjects and Controls:

Seventy newly diagnosed essential hypertensive patients attending the medicine OPD or admitted to the medical wards of Govt. Rajaji Hospital for the period of one year from February 2005 to January 2006 formed the study group. Thirty healthy people were kept as controls. This control group comprised of normotensive individuals who were attendants of patients with primary hypertension living in the same environment other than their own siblings.

Details of the Study Subjects:

All the patients were subjected to detailed history taking, careful physical examination and biochemical analysis to exclude secondary hypertension.

Patient's height and weight were measured. The body mass index was calculated using the formula weight / height ^{2}. Patient's hip and waist circumferences were measured. All the peripheral pulses were checked with special attention to carotid and the

femoral to detect evidence for early atherosclerosis. An ocular fundus examination was done to detect hypertensive retinopathy.

Patients were informed to refrain from smoking or drinking tea or coffee for at least thirty minutes before measuring blood pressure. Then blood pressure was measured using the following guidelines.

Guidelines for measuring blood pressure:

I CONDITIONS FOR THE PATIENT

A. Posture:

1. Sitting postures are usually adequate for routine follow-up. Patient should sit quietly with back supported for five minutes and arm supported at the level of heart.
2. For patients who are over 65, diabetic or receiving anti-hypertensive therapy, check for postural changes by taking readings immediately and 2 minutes after the patient stands.

B. Circumstances:

1. No caffeine for preceding hour
2. No smoking for preceding 15 minutes.
3. No exogenous adrenergic stimulants like phenylephrine in nasal decongestants or eye drops for papillary dilation.
4. A quite, warm setting.

5. Home readings taken under various circumstances and 24 hour ambulatory recordings may be preferable.

II EQUIPMENT:

A. Cuff Size:

The bladder should encircle and cover 2/3rds of the arm length. If not, place the bladder over the brachial artery; if bladder is small spuriously high readings may result.

B. Manometer:

1. Aneroid gauges should be calibrated every six months against the mercury manometer.
2. For infants use ultrasound equipment e.g., the Doppler method.

III TECHNIQUE:

A. Number of readings:

1. On each occasion, take at least two readings, separated by as much time as practical. If readings vary by more than 5 mm Hg, take additional readings until two are close.
2. For diagnosis, obtain at least three sets of readings a week apart.
3. Initially, take pressure in both arms, if pressure differs, use arm with higher pressure.

4. If arm pressure is elevated, take pressure in one leg, particularly in patients below age 30.

B. Performance:

1. Inflate the bladder quickly to a pressure 20 mm Hg above the systolic, as recognized by the disappearance of the radial pulse.
2. Deflate the bladder 3 mm Hg every second.
3. Record the Korotkoff phase V (disappearance) except in children, in whom use of phase IV (muffling) is advocated.
4. If Korotkoff sounds are weak, have the patients raise the arm, open and close the hand 5 to 10 times, after which the bladder should be inflated quickly.

C. Recording:

Note the pressure, patient position, the arm, cuff size (e.g., 140/90, seated, right arm, large adult cuff).

Urine albumin, sugar, microscopy and pH were done for all the subjects. A twelve lead electrocardiogram and chest x ray were also taken.

Overnight (12 hour) fasting blood sugar and urea was done by using Diacetyl monoxime (DAM) technique. Serum creatinine was estimated using COBAS auto

analyzer. Serum sodium and potassium was estimated using Flame emission photometric method.

DEFINITIONS USED IN THE PRESENT STUDY:

Essential Hypertension:

Hypertension was defined in accordance to the JNC- VII report as systolic blood pressure 140 mm of Hg and above and or diastolic blood pressure 90 mm of Hg and above. The diagnosis that the hypertension is essential and not secondary was made on the overall clinical impression only. Laboratory investigations to rule out secondary causes were not done in each case.

Sodium and Potassium Normal Values:

The normal range for serum sodium was from 135 to 150 mmol / L. The normal range for serum potassium was from 3.5 to 5 mmol / L.

Obesity:

According to the proposed classification of weight by BMI in adult Asians (55), the patients with a BMI <18.5 were classified as underweight, 1.85 – 22.9 were classified as normal, ≥ 23 were classified as overweight and ≥ 25 were classified as obese.

Diabetes Mellitus:

Patients with fasting plasma glucose ≥ 126 mg / dl or two hour plasma glucose ≥ 200 mg / dl or with symptoms of diabetes plus random blood glucose ≥ 200 mg / dl were considered to be diabetic.

Left ventricular hypertrophy:

Based on the electrocardiographic findings, which satisfy either Sokolow-Lyton criteria or Cornell voltage criteria (64, 65).

Conflict of interest:

There was no conflict of interest.

Financial Support:

Nil.

Limitations:

1. Only serum sodium and potassium were done.
2. Twenty four hours urinary sodium and potassium and arterial blood gas analysis were not done due to technical and financial limitations. Renal handling of sodium and potassium was not attempted as it was beyond the scope of the present study.
3. Body water content was not assessed which may alter the sodium and potassium levels.

4. Tissue sodium and potassium was not measured nor correlated with serum sodium and potassium levels.
5. Hormones related to sodium and potassium handling in kidney was not estimated.
6. The salt intake of the patients could not be assessed quantitatively and qualitatively because of social constraints.

Statistical Analysis:

The collected data was entered in Microsoft excel spread sheet and analysed statistically using epidemiological Information package – 2002 developed by centers for disease control and prevention, Atlanta in collaboration with World Health Organization. Student ‘t’ values were applied for significance. Significance was considered if the ‘p’ value was below 0.05.

RESULTS AND OBSERVATIONS

The total number of subjects included in this study was 100. Among these 100 subjects, 70 were cases (Hypertensive) and 30 were controls (Normotensive).

Analysis of cases and controls with respect to age:

The age of the subjects in the study group ranged from forty to sixty years. The mean and standard deviation for the age of the cases and controls were 53.1 ± 5.37 years and 51.5 ± 5.38 years respectively. The study group and the control group did not differ from each other statistically with reference to age.

The distribution of the cases and controls in relation to age is provided in the Table – IV given below (fig – 1).

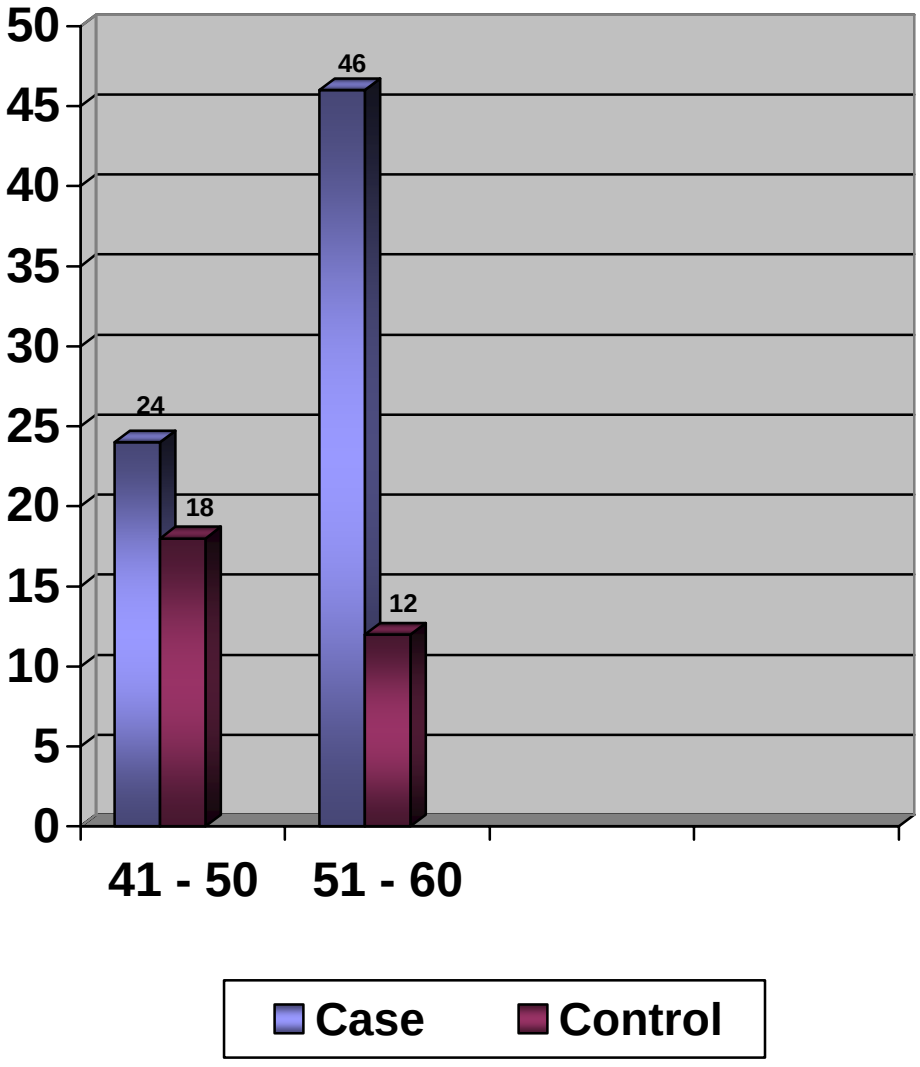
Table – IV

Distribution of cases and controls in relation to age

Age group	Cases		Controls	
	No	%	No	%
41 – 50	24	34.3	18	60
51 – 60	46	65.7	12	40
Total	70	100	30	100
Mean	53.1		51.5	
S.D	5.37		5.38	

Fig – 1

Distribution of cases and controls in relation to age



Majority of the patients in both the study and control group lie between 41 and 60 years. There was no significant difference in the age composition of those with and without hypertension in this study. Almost same age group of patients was selected in both groups.

The mean age distribution for the males in the case and control groups was 52.92 ± 5.52 years and 50.28 ± 5.66 years respectively. The mean age distribution for the females in the case and control groups was 53.31 ± 5.27 years and 51.8 ± 2.17 years respectively.

Gender:

Among the 70 cases studied, there were 38 males and 32 females. Among the 30 controls, there were 20 males and 10 females.

The details are given in the Table – V provided below and shown in fig - 2.

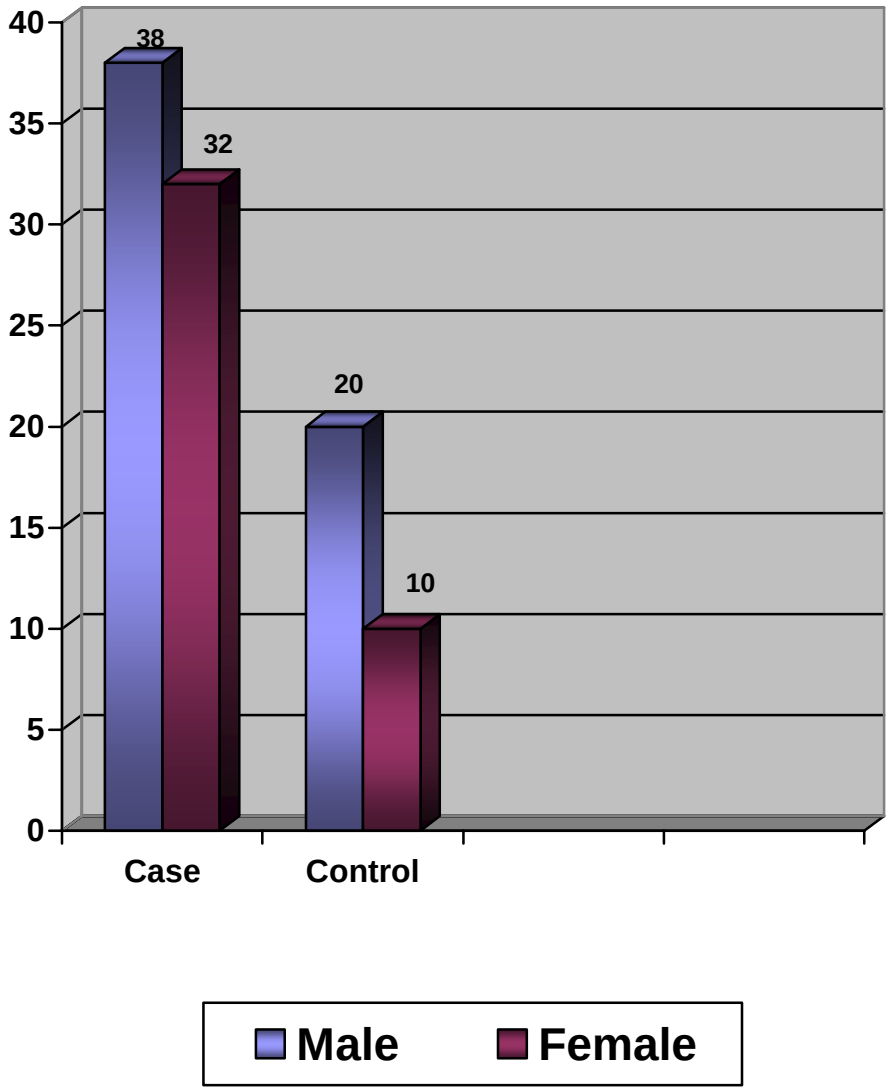
Table – V

Distribution of cases and controls in relation to gender

Sex	Cases		Controls	
	No	**%**	**No**	**%**
Male	38	54.3	20	66.7
Female	32	45.7	10	33.3
Total	70	100	30	100

fig – 2

Distribution of cases and controls in relation to gender



Analysis of cases and controls with respect to Body Mass Index (BMI)

34.3% of cases were obese while in the control group obesity was noticed in 3.3%. The details are shown in the Table – VI given below (fig – 3).

Table – VI

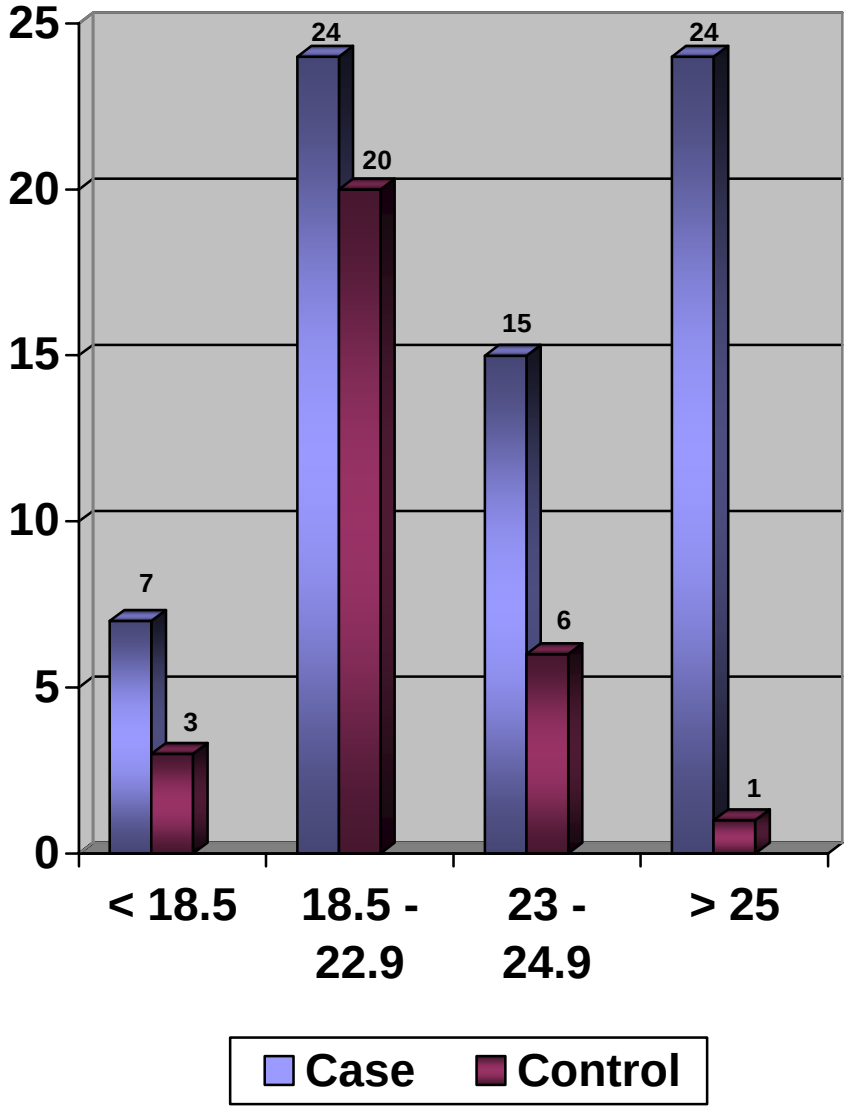
Distribution of cases and controls with respect to Body Mass Index (BMI)

BMI	Cases		controls	
	No	%	No	%
Underweight <18.5	7	10	3	10
Normal weight 18.5 – 22.9	24	34.3	20	66.7
Overweight 23 – 24.9	15	21.4	6	20
Obese > 25	24	34.3	1	3.3
Total	70	100	30	100

Fig – 3

Distribution of cases and controls with respect to

Body Mass Index (BMI)



The mean body mass index in the case group is 23.73 ± 3.28 and in the control group is 21.36 ± 2.12. The details are given in the Table – VII given below (fig – 4).

Table – VII

BMI among cases and controls

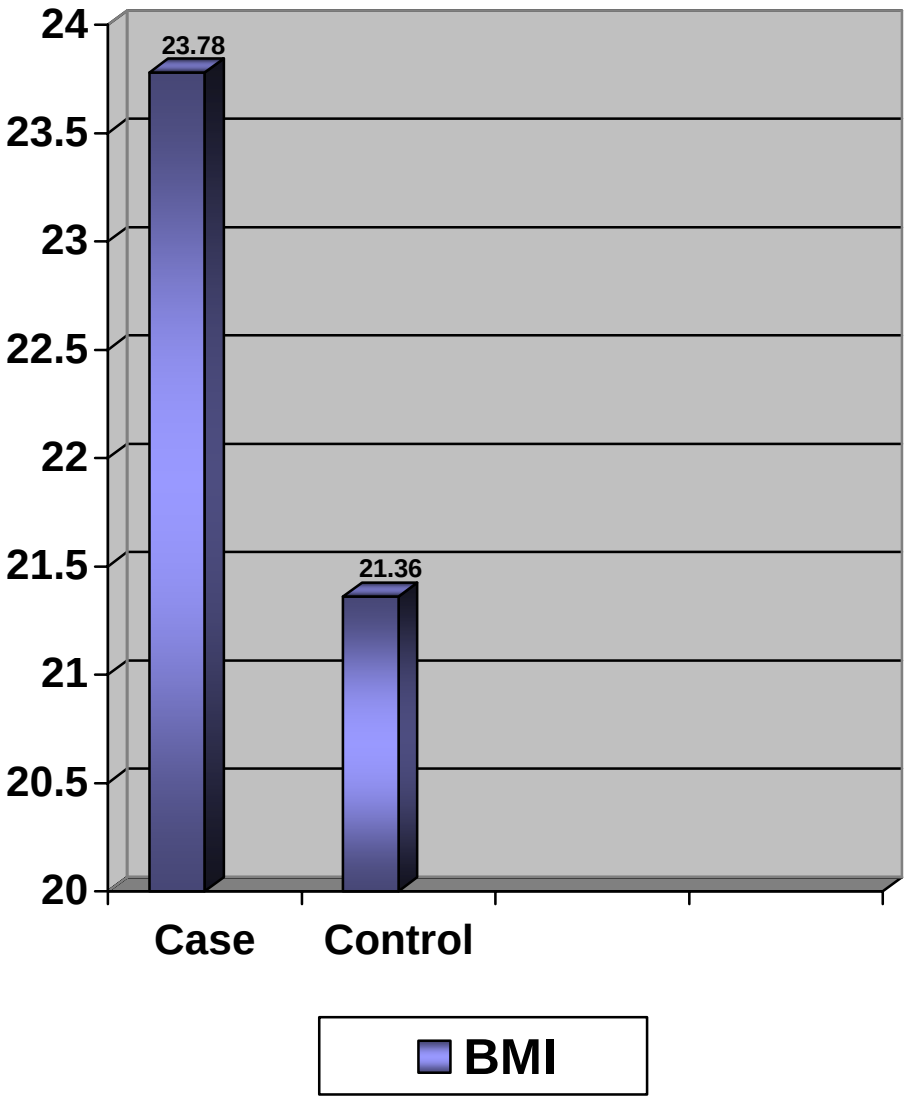
BMI	Cases	Controls
Mean	23.78	21.36
S.D.	3.28	2.12

'p' value = 0.00004

This shows that the difference in Body Mass Index between cases and controls was statistically significant.

Fig – 4

BMI among cases and controls



The mean BMI of cases and controls according to gender is given in the Table – VIII below (fig – 5).

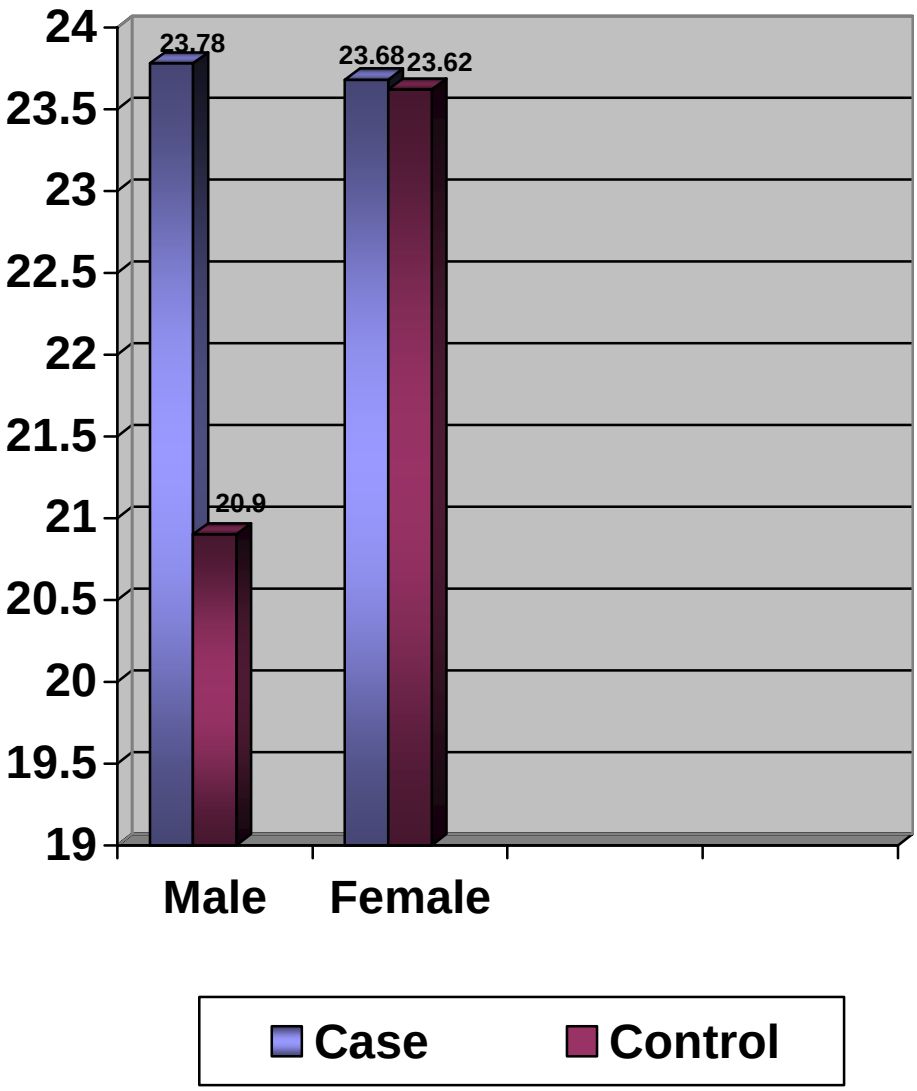
Table – VIII

BMI of cases and controls with respect to gender

Group	Male		Female	
	Mean	**S.D.**	**Mean**	**S.D**
Cases	23.78	3.49	23.68	3.07
Controls	20.9	1.62	23.62	2.21

Fig – 5

BMI of cases and controls with respect to gender



BMI in relation to the grading of hypertension is furnished in the Table – IX given below (fig – 6).

Table – IX

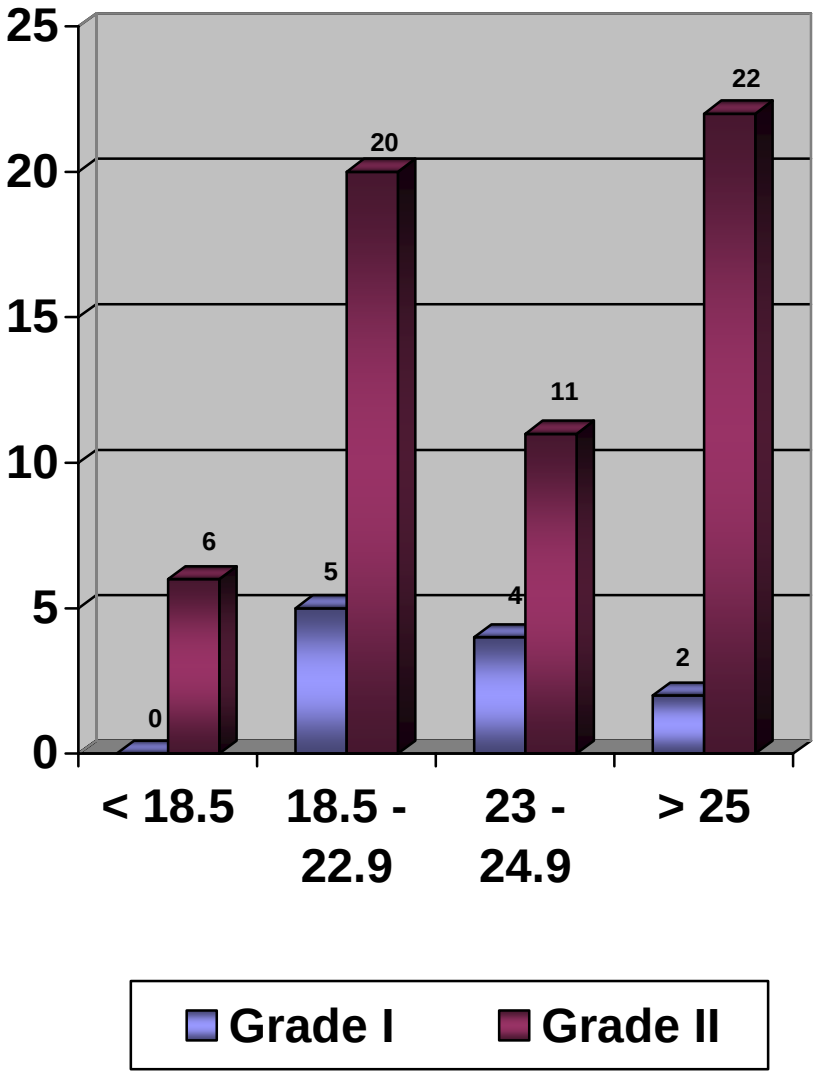
BMI with respect to Hypertension

BMI	Grade I Hypertension		Grade II Hypertension	
	No.	%	No.	%
Underweight <18.5	-	-	6	8.6
Normal 18.6 – 22.9	5	7.1	20	28.6
Overweight 23 – 24.9	4	5.7	11	15.7
Obese >25	2	2.9	22	31.4

Body mass index was independent of gender and electrolyte status, but it was significantly more in those with grade II hypertension.

Fig – 6

BMI with respect to Hypertension



Analysis of cases with respect to presenting symptoms

The most common presenting symptom is giddiness. The other symptoms were in the order of headache, chest pain, palpitation and dyspnoea. The details of the presenting symptoms are given in the Table – X given below (fig – 7).

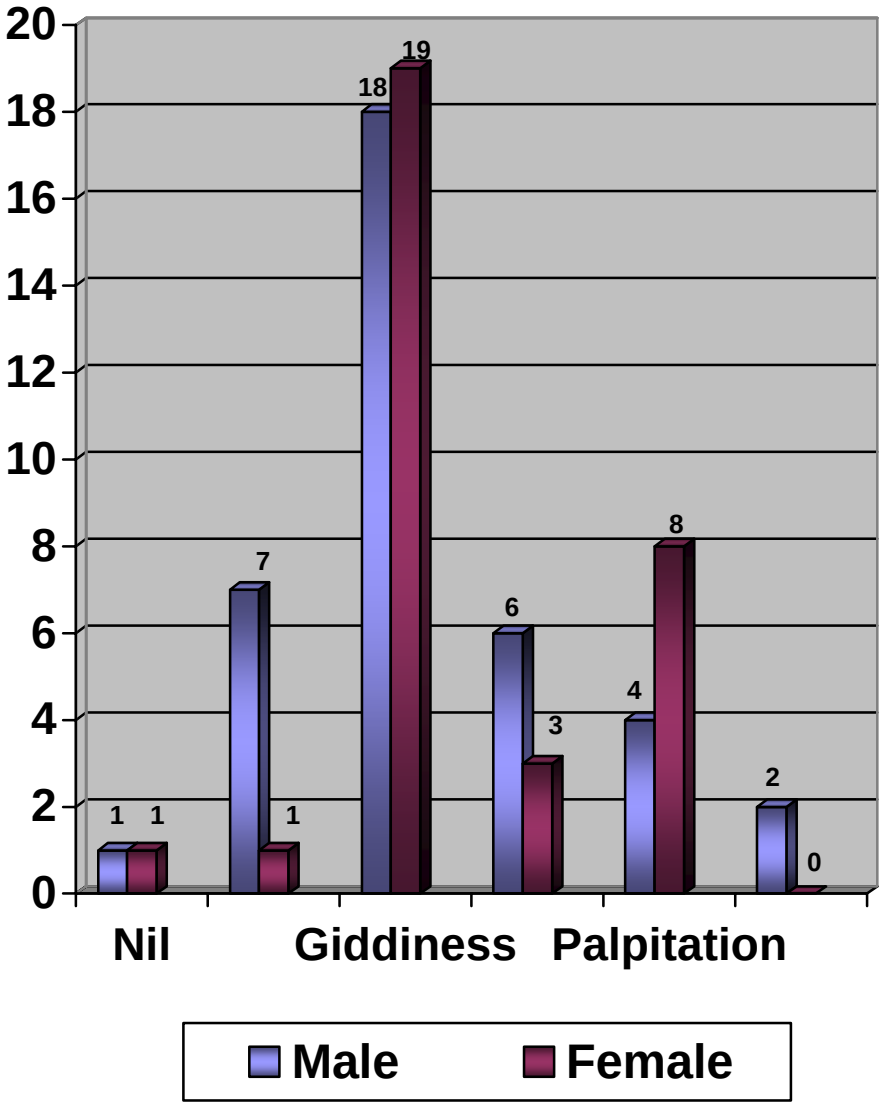
Table – X

Analysis of presenting symptoms

Symptoms	Male		Female	
	No.	%	No.	%
Nil	1	1.4	1	1.4
Headache	7	10	1	1.4
Giddiness	18	25.7	19	27.1
Chest pain	6	8.6	3	4.3
Palpitation	4	5.7	8	11.4
Dyspnoea	2	2.9	-	-

Fig – 7

Analysis of presenting symptoms



History of headache and chest pain was noticed among men and these patients were suffering from very high blood pressure. In contrast history of palpitation was elicited more among women.

Distribution of cases and controls with respect to cardio vascular risk factors

Analysis of other risk factors like smoking, alcoholism and family history were done among hypertensives. Their details are furnished in the Table – XI below.

Table – XI

Risk factors among cases and controls

	Smoking		**Alcohol**		**Both**	**Family history**	
	Yes	**No**	**Yes**	**No**		**Yes**	**No**
Cases	27	43	10	60	8	10	60
Control	14	16	6	24	8	-	30

Since alcoholism and smoking were noticed among men only in this part of the country, statistical analysis was not attempted for these risk factors.

Blood pressure distribution among cases

The details of the blood pressure distribution are given in the Table – XII given below (fig – 8).

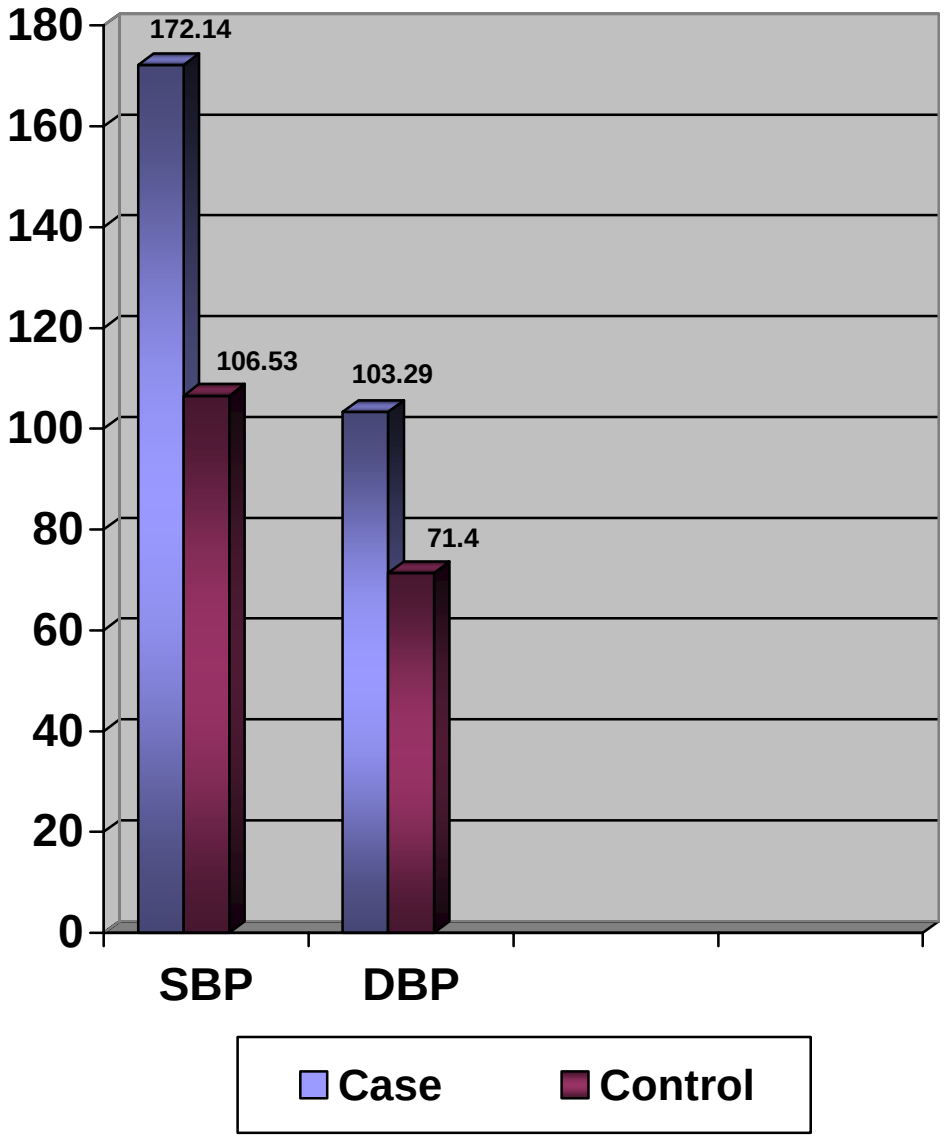
Table – XII

Distribution of systolic and diastolic blood pressure

Blood Pressure	Cases	Control
	Mean + SD	Mean + SD
Systolic	172.14 ± 15.12	106.53 ± 6.37
Diastolic	103.29 ± 6.07	71.4 ± 4.24

Fig – 8

Distribution of systolic and diastolic blood pressure



The mean systolic blood pressure for the cases was 172.14 ± 15.12 mm Hg. Similarly the mean diastolic blood pressure for the cases was 103.29 ± 6.07 mm Hg. Since the systolic and diastolic blood pressure was elevated in cases and it was due to the nature of the disease taken into study, the statistical analysis was not done.

The mean systolic and diastolic blood pressure distribution for the males was 172.63 ± 16.71 mm Hg and 103.42 ± 7.08 mm Hg respectively. Similarly for the females the mean systolic and diastolic blood pressure distribution was 171.56 ± 13.22 mm Hg and 103.13 ± 4.71 mm Hg respectively. There was no statistical significance in the systolic and diastolic blood pressure among the cases.

The distribution of cases with respect to grading of hypertension is given in the Table – XIII given below (fig – 9).

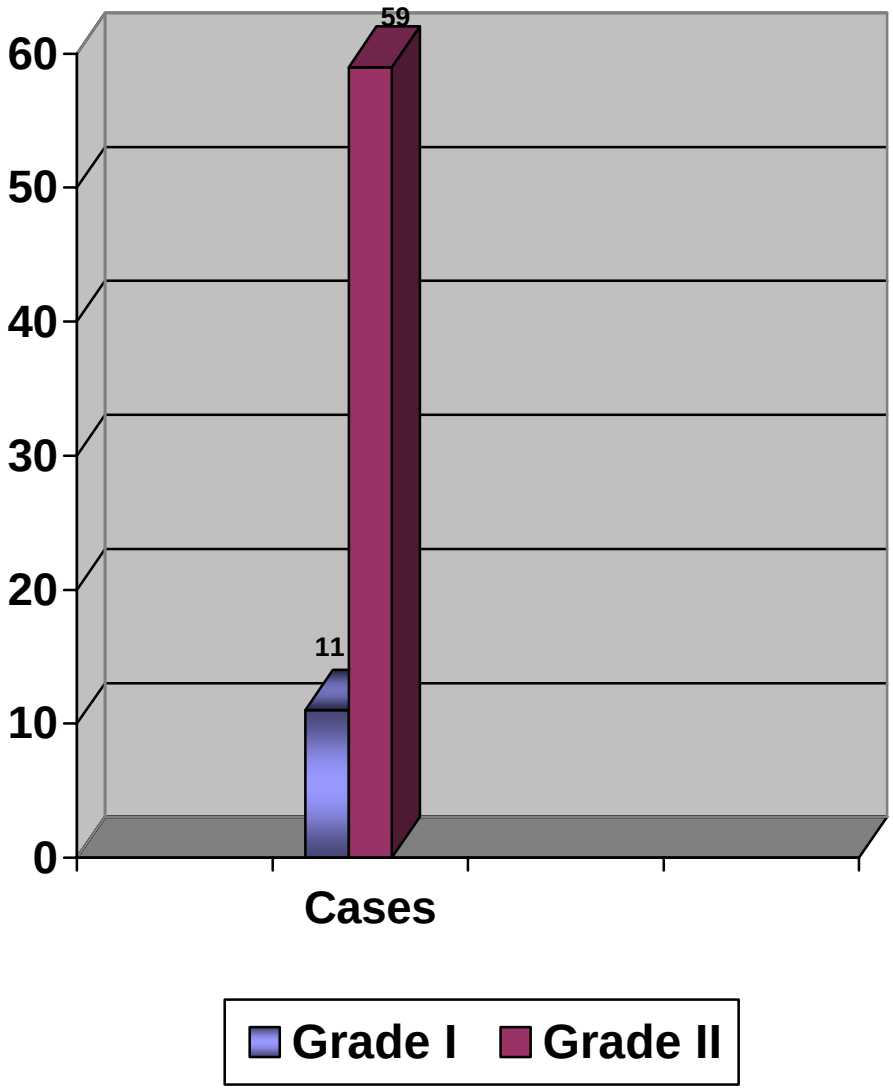
Table – XIII

Distribution of cases with respect to grading of hypertension

Subject	Grade I		Grade II	
	No	%	No	%
Cases	11	15.7	59	84.3

Fig – 9

Distribution of cases with respect to grading of hypertension



Distribution of cases and controls in relation to serum sodium

Serum sodium in the study population varied from 132 to 158 mmol / L and in the control from 136 to 146 mmol / L. The mean and standard deviation of serum sodium among cases was 145.41 ± 5.55 mmol / L while in the control group it was 139.9 ± 3.21 mmol / L respectively. This table clearly shows that the serum sodium level was significantly more among hypertensive population studied.

The details are shown in the Table – XIV given below (fig – 10).

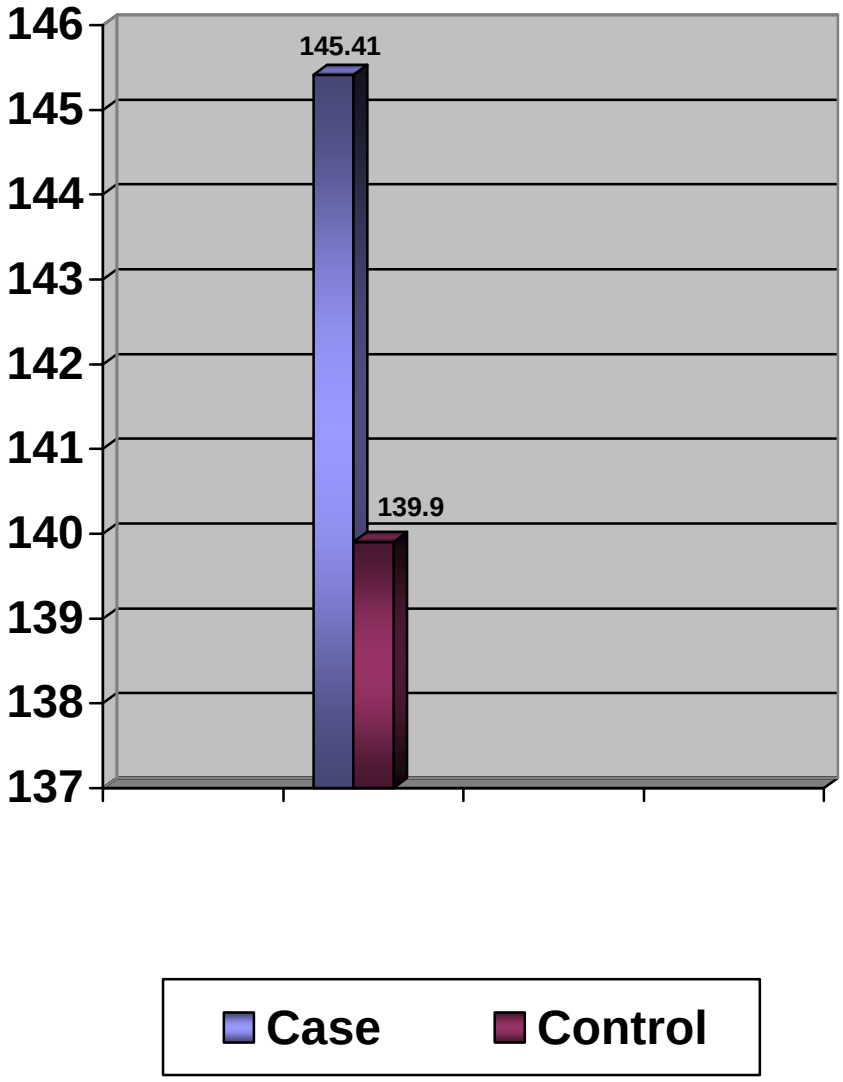
Table – XIV

Serum Sodium levels in cases and controls

Serum Sodium	Case		Control		'p' value
	Mean	SD	Mean	SD	
	145.41	5.55	139.9	3.21	0.000001

Fig – 10

Serum Sodium levels in cases and controls



Serum sodium in relation to gender

The mean value of serum sodium was 146.21 ± 4.81 mmol / L in males and 144.47 ± 6.27 mmol / L in females among cases. The mean value of serum sodium was 139.92 ± 3.38 mmol / L in males and 139.8 ± 2.49 mmol / L in females among controls. This is shown in Table – XV given below (fig – 11).

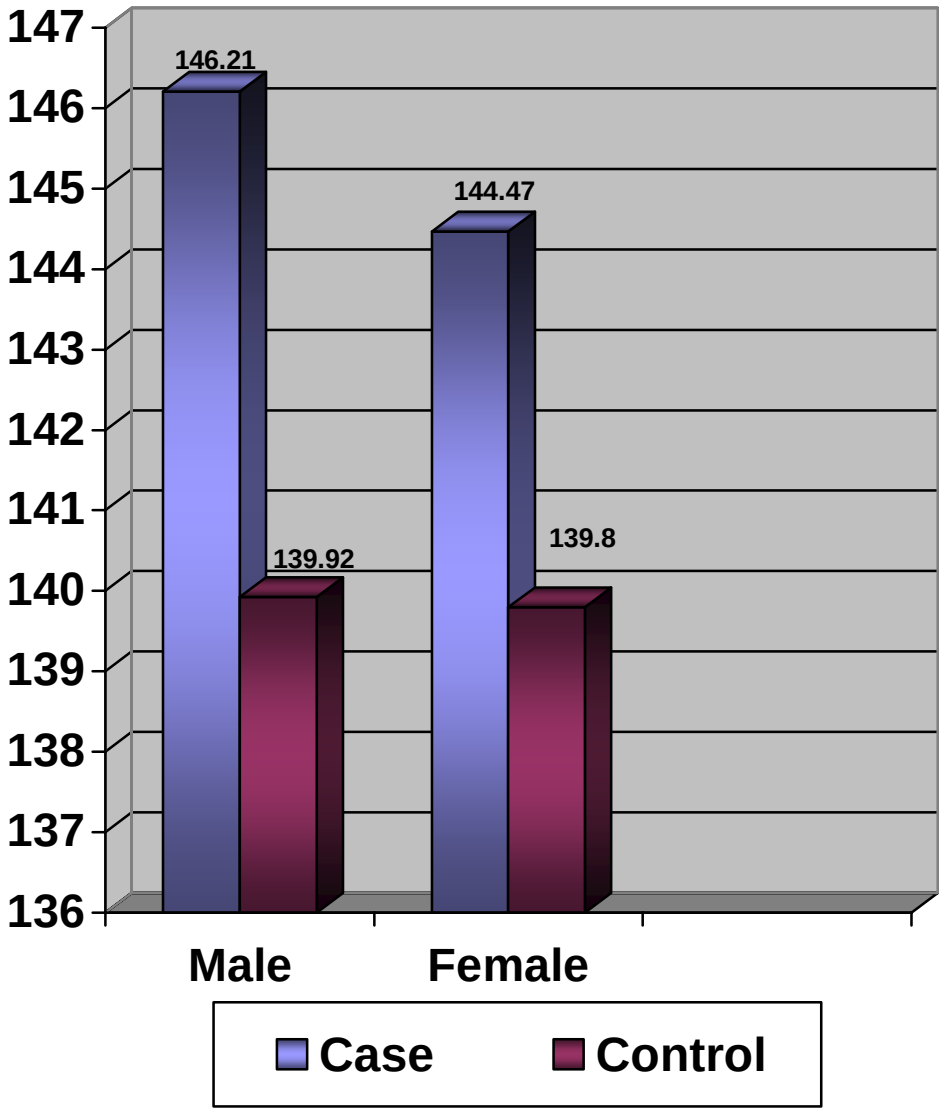
Table – XV

Serum Sodium values in relation to gender

Sex	Case	Control
Male	146.21	139.92
Female	144.47	139.8

Fig – 11

Serum Sodium values in relation to gender



The mean serum sodium level though appeared to be more among males, the difference among males and females was not significant statistically.

Distribution of cases and controls in relation to serum potassium

Serum potassium in the study population varied from 3.1 to 5.2 mmol / L and in the control from 3.8 to 4.8 mmol / L. The mean and standard deviation of serum potassium among cases was 4.03 ± 0.49 mmol / L while in the control group it was 4.29 ± 0.33 mmol / L respectively. This table clearly shows that the serum potassium level was significantly lower among the hypertensive population studied.

The details are given in Table – XVI given below (fig – 12).

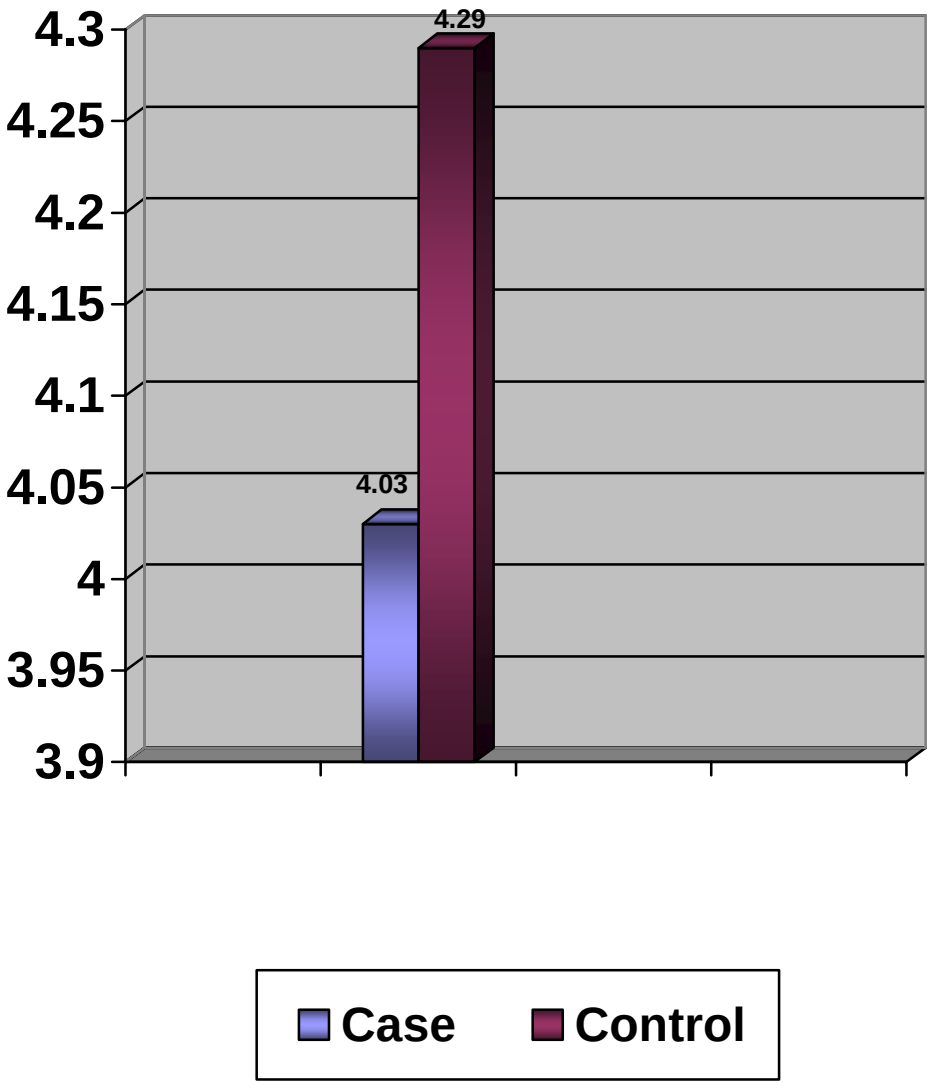
Table – XVI

Serum potassium level in cases and controls

Serum Potassium	Case		Control		'p' value
	Mean	S.D	Mean	S.D	
	4.03	0.49	4.29	0.33	0.0026

Fig – 12

Serum potassium level in cases and controls



Serum potassium in relation to gender

The mean value of serum potassium was 4.08 ± 0.49 mmol / L in males and 3.98 ± 0.50 mmol / L in females among cases. The mean value of serum potassium was 4.26 ± 0.32 mmol / L in males and 4.22 ± 0.40 mmol / L in females among controls. This is shown in Table – XVII given below (fig -13).

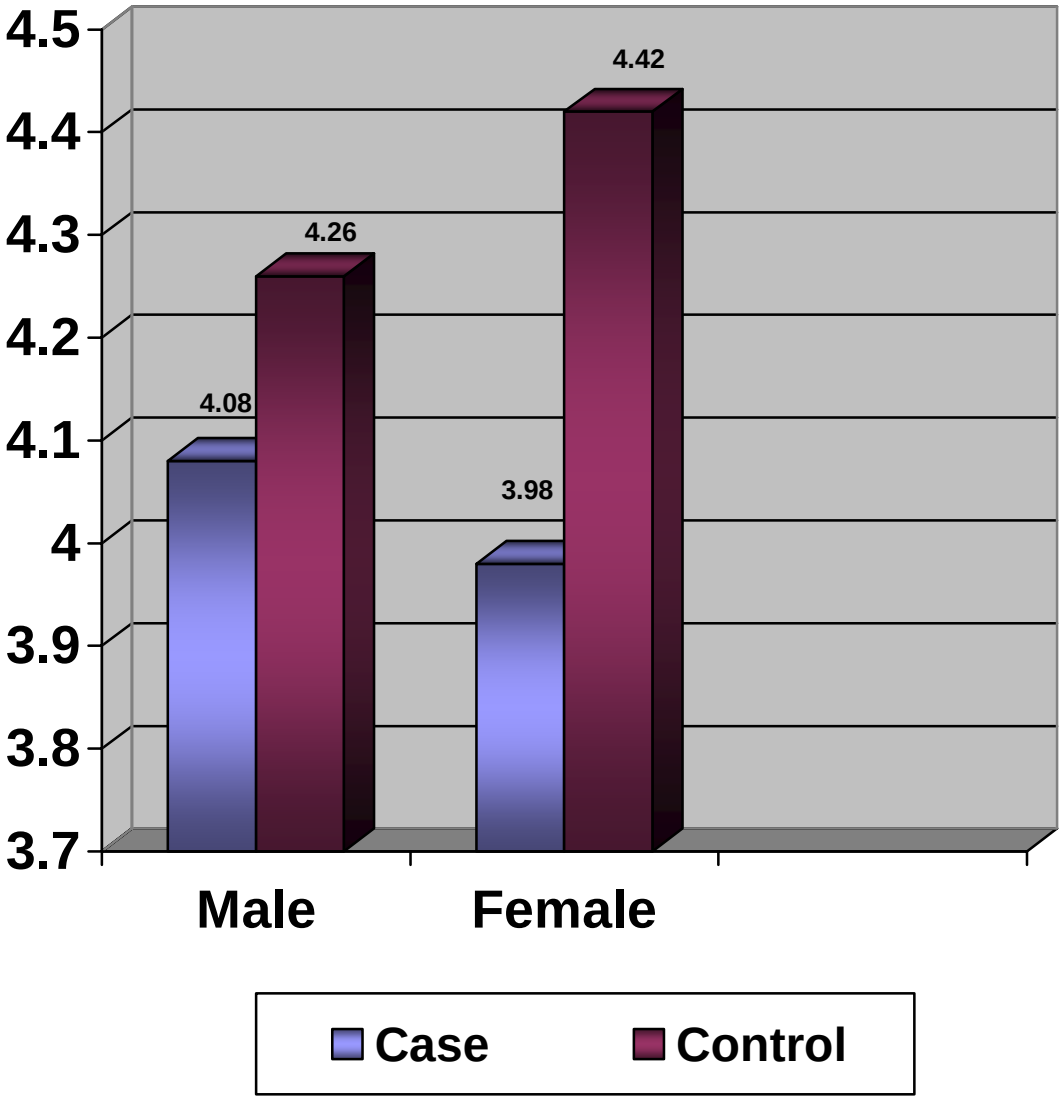
Table – XVII

Serum potassium values in relation to gender

Sex	Cases	Controls
Male	4.08	4.26
Female	3.98	4.22

Fig – 13

Serum potassium values in relation to gender



The mean serum potassium level though appeared to be more among males, the difference among males and females was not significant statistically.

Urinary analysis was not contributory. Blood glucose, blood urea and serum creatinine were well within acceptable limits and did not differ from healthy control.

Electrocardiogram revealed left ventricular hypertrophy in about 20 percent of study group. Chest X ray showed cardiomegaly in about 16 percent of cases only. Ocular fundus examination revealed hypertensive retinopathy in about 38 percent of the study group.

DISCUSSION

Hypertension is one of the leading causes of death and disability among adults all over the world. Hypertension the most common form of cardiovascular disease is present in nearly 25% of adults and increases in prevalence with age. It remains the major risk factor for coronary, cerebral and peripheral vascular disease. Essential hypertension comprises more than 90% of hypertension (1).

Patients were studied on the basis of clinical parameters and simple biochemical investigations. Serum sodium and potassium was done for all the patients.

Serum sodium among Hypertensives:

In our part of the country, there is excessive intake of dietary salt. But in spite of that not everyone has essential hypertension. The rarity of hypertension among those consuming large amount of salt may probably be related to chronic adaptation of body system towards renal clearance of sodium. However this aspect of chronic adaptation of sodium handling by kidneys requires further molecular studies. So in addition to the hereditary predisposition and high sodium intake and lower potassium intake, the renal handling of these cations also play an important role in the pathogenesis of essential hypertension (58-60).

Salt intake was more in the tropical countries by and large in order to overcome sodium loss through sweating. In modern days the consumption of salt is more than

earlier days in view of various food preparations or a combination of them, as man is tuned more to taste of the food. Combination of food materials requires additional salt. As a result, people consume more than actually required (2 vs. 8-10 g / day / person). Such an amount of salt consumption contributes for the development of hypertension in a genetically susceptible population.

In our study the mean serum sodium was estimated in the control and study groups. Results were compared with other studies.

Serum sodium was higher in the hypertensive group than the control group even though both were within the normal range. The mean and standard deviation of serum sodium among cases was 145.41 ± 5.55 while in the control group it was 139.9 ± 3.21 respectively.

Our study was supported by Jan et al (2006), Srinagar, Kashmir. In his study, one hundred thirty five hypertensive patients and equal number of age and sex matched healthy controls were taken for the study. Serum sodium in the hypertensive group was 140 ± 2.90 while in the control group it was found to be 138.5 ± 1.12. Serum sodium was higher in the hypertensive group than the control group and considered to be a factor responsible for the causation or perpetuation of blood pressure (66).

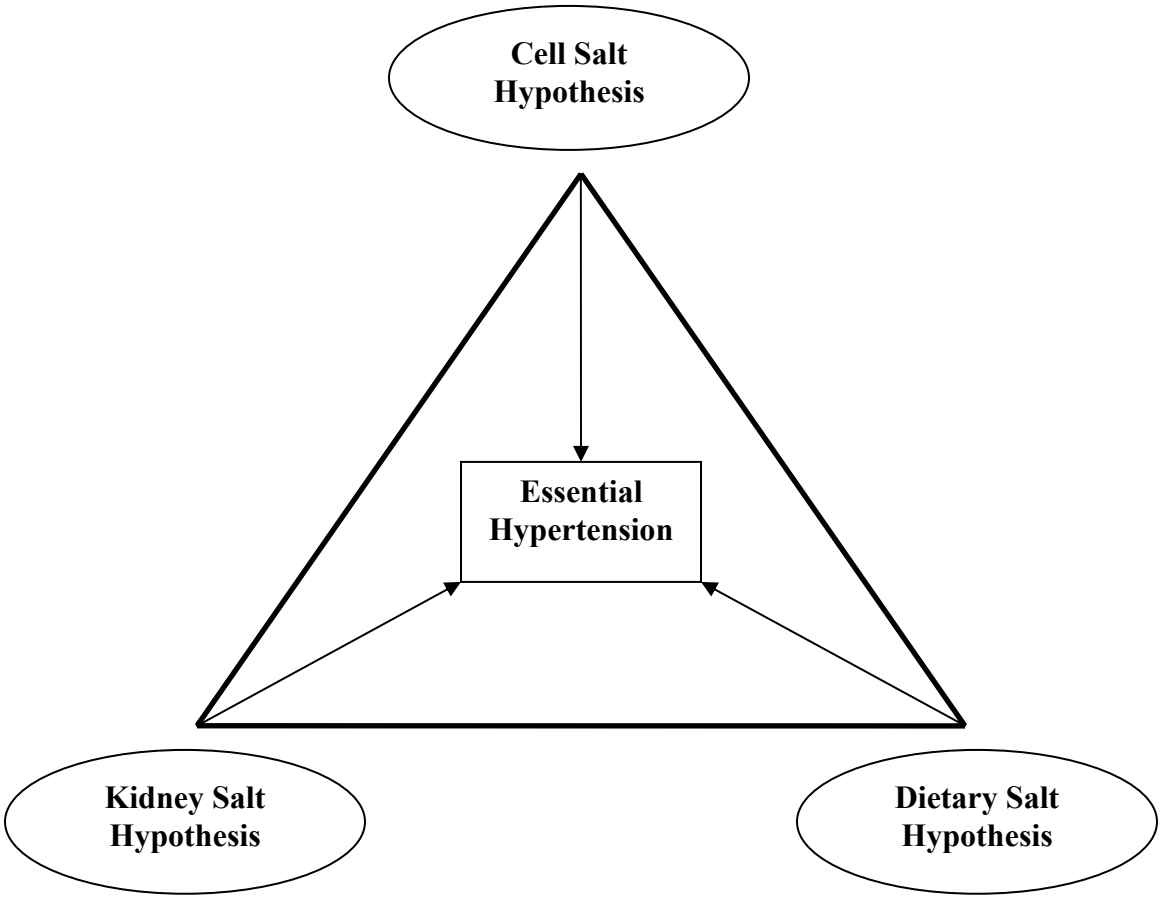
A study was carried out by Lever et al of arterial pressure and body content of electrolytes in 91 patients with essential hypertension and 121 normal controls (67).

Plasma and exchangeable sodium was found to be positively correlated with arterial pressure in the patients. Three hypotheses were proposed to explain the mechanisms relating electrolytes and arterial pressure in essential hypertension and it was depicted in pictorial form in figure – 14 shown in the following page.

1. Cell-salt hypothesis
2. Dietary salt hypothesis
3. Kidney-salt hypothesis.

Figure - 14

Proposed hypotheses by Lever et al in the pathogenesis of essential hypertension



It was concluded that two mechanisms probably operate in essential hypertension. In the early stages of the disease, blood pressure is raised by an abnormal process related more closely to potassium than to sodium. A renal lesion develops later, possibly as a consequence of the hypertension. This lesion is characterised by resetting of pressure natriuresis and is manifest by an abnormal relation between body sodium and arterial pressure and by susceptibility to increased dietary sodium intake.

In another study conducted by Williams et al, they studied the relationship of body sodium, chlorine and potassium in 30 patients with essential hypertension. They found that a positive correlation exists between serum sodium and blood pressure in this study group (68).

In another study conducted by Nanji et al, it was shown that a positive correlation exists between serum sodium and hypertension (69).

A study was conducted among Japanese people by Komiya et al. They studied 3222 normal Japanese subjects (610 in Kashiwa City Hospital and 2612 in Shinshu University Hospital), 741 Japanese patients with essential hypertension (256 in Kashiwa City Hospital and 485 in Shinshu University Hospital) to determine the possible roles of sodium, renal function, and plasma aldosterone concentration (PAC) on blood pressure elevation.. They found that the peak of the serum sodium distribution curve was approximately 2 mmol / L higher in the hypertensive group as compared with that in the control group. The prevalence of higher serum sodium concentration (> or = 147 mmol/l) was also significantly higher in the hypertensive group (70).

In another study conducted by Bulpitt, two thousand, three hundred and twenty-eight men and 1496 women between the ages of 35 and 64 years were screened for hypertension and their plasma sodium and potassium concentrations measured. It was found that plasma sodium was positively related to that of blood pressure and an increase in serum sodium of 1 mmol / L was associated with an increase of 1 mm of Hg in both men and women (71).

Serum potassium among Hypertensives:

In our study serum potassium was estimated in control and study groups and compared between them. Serum potassium was found to be lower in the hypertensive group when compared with the control group even though both were within the normal range.

The mean serum potassium in the study group was 4.03 ± 0.49. The mean potassium in the control group was 4.29 ± 0.33.

A study was carried out by Bulpitt et al among two thousand, three hundred and twenty-eight men and 1496 women between the ages of 35 and 64 years and were screened for hypertension and their plasma sodium and potassium concentrations measured. Those on antihypertensive or diuretic treatment were excluded from further analysis. After adjusting for age, body mass index and other variables, plasma potassium was negatively associated with both systolic and diastolic pressure in men and women. A decrease in plasma potassium of 1 mmol/l was associated with an increase in systolic

pressure in women of 7 mmHg (P less than 0.001) and diastolic pressure of 4 mmHg (P less than 0.001). In men the corresponding increases were 4 mmHg (P less than 0.01) and 2 mmHg (P less than 0.05) (71).

Similarly in a study conducted by Lever et al in 121 normal subjects and 91 hypertensive patients, it was shown that plasma, exchangeable, and total body potassium correlated inversely with arterial pressure in the patients. They have suggested the following theory as the cause of essential hypertension. In the early stages of the disease blood pressure is raised by an abnormal process related more closely to potassium than to sodium. A renal lesion develops later, possibly as a consequence of the hypertension. This lesion is characterised by resetting of pressure natriuresis and is manifest by an abnormal relation between body sodium and arterial pressure and by susceptibility to increased dietary sodium intake (67).

Similarly a study was conducted at National Institute of Public Health and Environmental Protection, Bilthoven, The Netherlands. The relationships between the serum cations sodium, potassium, calcium and magnesium and blood pressure were investigated in a population-based sample of 182 Dutch persons aged 20-59 years. In the combined analysis, a weak inverse relationship was found between serum potassium and diastolic blood pressure; this relationship was also found in women (72).

In an another study carried out at Karachi, Pakistan, thirty hypertensive diabetic patients and equal number of age and sex matched controls were taken for the study. The

mean serum potassium was 4.59 mmol / l among the study group while 5.03 mmol / l among the control group (73).

In a study carried out at the University of Tokyo, they measured plasma electrolytes in 82 essential hypertensive patients to examine the relation between blood pressure and plasma electrolytes. Significant negative correlations were observed between plasma potassium concentration and 24-h systolic blood pressure (r = -0.336) and diastolic blood pressure (r = -0.298) in their patients. Plasma potassium concentration inversely correlated also with both daytime and nighttime systolic and diastolic blood pressure. There was no relation between office blood pressure and plasma potassium concentration. These findings indicate that in essential hypertensives plasma potassium concentration is inversely related to ambulatory blood pressure including daytime and nighttime blood pressure and suggest that potassium may be a factor determining the whole day blood pressure in essential hypertension (74).

To investigate the role of potassium on blood pressure Luft et al, conducted a study among 431normotensive and 478 hypertensive subjects. They observed an inverse relationship between serum potassium and blood pressure supporting our study (75).

BMI and Hypertension:

In our study the mean BMI among the study group was 23.73 ± 3.28 and among the control group was 21.36 ± 2.12. The 'p' value was .00004. This shows that overweight and obesity also plays a role in the development of essential hypertension.

This was supported by a study conducted by Stamler (54). They showed that the hypertension is about six times more common in obese than it is in lean subjects. The present study concurs with above observation. However body mass index was not related to electrolyte levels.

Similarly a study conducted by Huang stated that even a small amount of weight gain is associated with a marked increase in the incidence of hypertension (76). This study showed a positive correlation between BMI and blood pressure which supported our study.

In INTERSALT, the relationship between body mass index (kg/m2) and blood pressure was studied in 10,079 men and women aged 20-59, sampled from 52 centres around the world, based on a standardized protocol with central training of observers, a central laboratory and extensive quality control. Body mass index-blood pressure relationships were first studied in men and women within each centre, and results of these regression analyses were then pooled for all 52 centres. With adjustment for age, alcohol intake, smoking, and sodium and potassium excretion, body mass index was positively associated with systolic blood pressure among men in 51 of 52 centres and among women in 47, significantly so in 24 and 27, respectively. Body mass index was positively associated with diastolic blood pressure in 51 and 49 centres in men and women, respectively, significantly so in 33 and 31. Overall, a 10 kg difference in body weight was associated on average with a 3.0 mmHg difference in systolic and a 2.2 mmHg difference in diastolic pressure. In further analyses across centres, median body mass index was

related significantly to median systolic blood pressure, median diastolic pressure and the prevalence of hypertension in both men and women. Body mass index was related to the slopes of systolic and diastolic blood pressure with age in women, but not in men (77).

Areas for further research

1. To identify the markers of hypertension prone population.
2. To find out the genes responsible for renal sodium handling and if required to develop a biomolecule to overcome the effects of the mediators of which signal / control the sodium and potassium at the molecular level.

CONCLUSION

The following conclusions were derived from our study.

1. Serum sodium was significantly more among hypertensives and it was independent of associated risk factors and gender.

2. Serum sodium level was also correlated positively with the level of blood pressure.

3. Serum potassium was significantly less among hypertensives and it correlated negatively with blood pressure.

4. Serum sodium and potassium were independent of body mass index.

5. In view of the significant changes in simple electrolyte levels (sodium and potassium) among hypertensive population, community must be motivated to reduce their intake of common salt and encouraged to consume potassium rich nutrients – diets as a form of primary prevention for essential hypertension.

SUMMARY

Essential hypertension is the major risk factor for coronary, cerebral and renal vascular diseases. Etiology for essential hypertension is not known. Many theories were postulated.

The present study attempts to focus the serum sodium and potassium level among isolated newly diagnosed essential hypertensives who were free from any other illnesses or under any medication and to correlate electrolyte status with the blood pressure. Fasting serum sodium and potassium were estimated (flame photometer) in seventy hypertensives (m=38, f=32; mean age 53.1 ± 5.37) and thirty healthy controls (m=20 f=10; mean age 51.5 ± 5.38). Efforts were also made to find out an association between body mass index and waist circumference with systolic and diastolic blood pressure. Body mass index was significantly more in those with stage II hypertension. However it was independent of gender and electrolyte status. Mean serum sodium level was elevated significantly ('p' = 0.000001) among hypertensives where as serum potassium level was significantly lower among them when compared to healthy controls. The blood pressure also correlated positively with serum sodium; body mass index and waist circumference where as negatively correlated with serum potassium.

Changing life styles have modified the food habits, making people to consume food rich in sodium but low in potassium. As a result genetically susceptible population

when exposed to high sodium content coupled with low potassium in their diet, hypertension becomes overt. The possible mechanisms were discussed.

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PROFORMA

Name: Age: Sex: M / F Diet: V / NV
Address: Occupation:

SYMPTOMS:

□ Headache	□ Oliguria	□ Giddiness
□ Puffiness of face	□ Blurring of vision	□ Epistaxis
□ Swelling of legs	□ Anorexia	□ Chest pain
□ Vomiting / Hiccups	□ Palpitation	□ Easy fatiguability
□ Dyspnoea	□ Polydipsia	□ Polyuria

PAST HISTORY

□ DM	□ Angina / MI	□ Heart Failure
□ Renal Disorders	□ PVD	□ Stroke

PERSONAL HISTORY

□ Smoking	□ Alcohol	□ Drug abuse

FAMILY HISTORY

□ Hypertension	□ Diabetes Mellitus

ANTHRPOMETRY

Ht	cm	Wt	kg	BMI
Hip	cm	waist	cm	WHR

GENERAL EXAMINATION

Fundus	Blood pressure	
Pedal edema	Supine	Sitting
Pulse Rate	Grading of Hypertension	

SYSTEMIC EXAMINATION

CVS	RS
ABD	CNS

INVESTIGATIONS

Urine Albumin
Sugar
Deposits

Blood Glucose
Urea
Serum Creatinine

ECG

CXR PA VIEW

Serum Sodium
Potassium

MASTER CHART

Sl. No	Age / Sex	Symptoms	P / H	Smoking	Alcohol	F / H	BMI	WHR	Fundus	SBP	DBP	SE	UR	Bl. Glucose	Bl. Urea	Sr. Creatinine	Sr. Sodium	Sr. Potassium	ECG	CXR
1	60/M	2	N	Y	No	N	17.7	0.88	N	160	100	N	N	78	20	0.7	140	3.6	N	N
2	52/M	2	N	Y	Y	N	25.7	0.99	N	190	120	N	N	92	26	1	148	3.5	L	C
3	56/M	5	N	No	No	N	27.5	1	N	190	120	N	N	68	20	0.7	146	3.6	B	N
4	55/M	2	N	Y	Y	N	17.9	0.9	I	180	110	N	N	114	24	0.9	150	3.7	N	N
5	60/M	5	N	No	No	N	24.8	1	N	200	110	N	N	128	25	1.2	145	3.8	L	C
6	45/F	2	N	No	No	N	19.5	0.8	N	160	100	N	N	80	32	1	149	3.6	N	N
7	50/M	4	D	Y	Y	N	25.4	0.9	I	170	90	N	N	92	30	0.9	147	4.9	B	N
8	45/F	2	N	No	No	N	28.7	0.83	I	190	110	N	N	100	18	0.9	137	3.8	N	N
9	55/F	2	N	No	No	N	28.8	0.9	N	180	100	N	N	88	18	0.8	141	3.4	N	N
10	46/M	1	N	No	No	H	29.7	0.96	N	150	100	N	N	77	17	1.2	155	3.4	N	N
11	47/M	2	N	No	No	H	23.1	0.9	N	150	100	N	N	107	26	107	148	3.2	N	N
12	56/M	2	N	Y	No	N	18.3	0.93	I	190	90	N	N	79	31	1.2	142	4.4	N	N
13	56/M	3	N	No	No	N	31.2	0.93	N	160	100	N	N	139	22	0.9	140	4.3	L	C
14	50/F	2	N	No	No	N	27.3	0.94	N	200	110	N	N	60	21	0.9	146	4.5	L	C
15	55/M	2	N	Y	No	N	22.8	0.94	I	150	100	N	N	61	29	1.2	138	4	N	N
16	59/M	1	N	Y	No	H	27.7	0.95	I	160	100	N	N	112	28	0.9	153	3.9	N	N
17	55/M	3	N	No	No	N	19.4	0.9	N	160	100	N	N	64	22	0.9	154	4	N	N

Sl. No	Age / Sex	Symptoms	P / H	Smoking	Alcohol	F / H	BMI	WHR	Fundus	SBP	DBP	SE	UR	Bl. Glucose	Bl. Urea	Sr. Creatinine	Sr. Sodium	Sr. Potassium	ECG	CXR
18	60/F	3	N	No	No	N	18.3	0.91	I	170	100	N	N	85	19	0.9	155	3.8	N	N
19	48/F	2	N	No	No	N	23.9	0.83	N	160	100	N	N	68	40	1.2	142	4.8	N	N
20	52/M	2	N	Y	Y	N	22.6	0.93	I	200	120	N	N	90	30	1.1	146	4.5	N	N
21	42/M	1	N	No	Y	H	19.7	0.92	N	150	100	N	N	85	20	0.8	158	3.8	N	N
22	54/F	2	N	No	No	N	24.9	0.83	I	190	110	N	N	105	38	0.9	142	4.5	N	N
23	46/M	1	N	No	No	N	25.7	0.95	N	150	110	N	N	108	18	1	148	4.3	N	N
24	50/M	2	N	Y	No	N	22.7	0.98	I	170	100	N	N	133	23	1	142	4.2	L	C
25	45/M	2	N	Y	No	N	23.4	0.9	N	150	100	N	N	80	24	1.2	145	4.8	N	N
26	50/F	2	N	No	No	N	21.4	0.88	I	160	110	N	N	60	28	0.8	153	4.1	N	N
27	56/F	0	N	No	No	N	19.5	0.9	N	160	100	N	N	115	31	1.1	146	3.4	N	N
28	60/F	1	N	No	No	N	21.7	0.9	N	170	110	N	N	92	20	0.8	152	3.5	N	N
29	60/F	2	N	No	No	H	23.2	0.85	N	170	100	N	N	84	22	0.8	148	3.5	N	N
30	60/M	0	N	Y	Y	N	20.1	0.85	N	180	100	N	N	108	27	1.1	141	4.6	N	N
31	60/F	2	N	No	No	N	17.9	0.98	I	170	100	N	N	66	41	1.1	144	3.6	L	C
32	53/F	2	N	No	No	N	22.8	0.78	N	170	100	N	N	84	20	0.6	155	3.9	N	N
33	45/M	2	N	Y	Y	H	20.7	0.93	N	160	100	N	N	83	27	0.8	150	3.6	N	N
34	59/F	2	N	No	No	N	22.8	0.8	N	160	100	N	N	71	23	0.6	144	4.5	B	N

Sl. No	Age / Sex	Symptoms	P / H	Smoking	Alcohol	F / H	BMI	WHR	Fundus	SBP	DBP	SE	UR	Bl. Glucose	Bl. Urea	Sr. Creatinine	Sr. Sodium	Sr. Potassium	ECG	CXR
35	43/F	2	N	No	No	N	24.3	0.8	N	150	100	N	N	72	20	0.7	148	4	N	N
36	56/M	1	N	No	No	N	27.5	1	N	190	110	N	N	68	20	0.7	148	3.9	B	N
37	55/M	2	N	Y	Y	N	21.7	0.91	I	190	110	N	N	124	24	0.9	150	4	N	N
38	60/M	2	N	No	No	N	24.8	1	I	190	100	N	N	131	25	1.3	146	4.2	N	N
39	48/F	2	N	No	No	N	24	0.82	N	180	100	N	N	84	32	1.1	148	4.2	N	N
40	54/M	3	N	Y	No	N	26.4	0.95	I	180	110	N	N	90	38	0.9	147	4.3	B	N
41	45/F	2	N	No	No	N	29.2	0.91	I	190	100	N	N	102	18	0.9	137	3.8	N	N
42	55/F	4	N	No	No	N	28.8	0.95	N	160	100	N	N	88	22	0.8	148	3.6	N	N
43	46/M	2	N	Y	No	N	29.3	0.96	N	170	100	N	N	92	22	1.1	145	3.2	N	N
44	52/F	2	N	No	No	H	25.1	0.9	N	160	100	N	N	107	26	1.1	148	3.1	L	C
45	52/M	2	N	Y	No	N	22.3	0.93	I	180	100	N	N	76	35	1.2	142	4.8	N	N
46	56/M	3	N	Y	No	N	29.9	0.93	N	170	100	N	N	126	22	0.8	142	3.8	L	C
47	50/F	2	N	No	No	N	27.3	0.94	I	190	110	N	N	96	28	0.9	146	4.5	L	C
48	55/M	1	N	No	No	N	22.8	0.94	N	150	100	N	N	116	40	1.2	138	4	N	N
49	48/M	1	N	Y	No	N	27.7	0.95	I	160	100	N	N	130	33	0.8	150	4.1	N	N
50	60/F	1	N	No	No	N	23.8	0.89	I	180	110	N	N	114	28	1.2	148	3.6	N	N
51	54/F	1	N	No	No	H	21.7	0.91	N	180	100	N	N	114	29	1	142	4	N	N

Sl. No	Age / Sex	Symptoms	P / H	Smoking	Alcohol	F / H	BMI	WHR	Fundus	SBP	DBP	SE	UR	Bl. Glucose	Bl. Urea	Sr. Creatinine	Sr. Sodium	Sr. Potassium	ECG	CXR
52	51/F	4	N	No	No	N	23.9	0.83	N	150	100	N	N	108	42	0.9	146	4.3	N	N
53	60/M	2	N	Y	No	N	22.9	0.93	I	190	110	N	N	96	32	1.1	140	4.5	L	C
54	41/M	3	N	Y	Y	H	21.7	0.92	N	150	100	N	N	84	26	0.9	150	3.4	N	N
55	49/F	2	N	No	No	N	25.1	0.83	I	190	110	N	N	105	28	0.9	142	4.5	N	N
56	49/M	1	N	Y	No	N	25.5	0.95	N	170	100	N	N	108	18	1	148	4.3	N	N
57	56/M	2	N	Y	No	N	21.4	0.98	I	170	100	N	N	133	23	1	142	5.2	L	N
58	48/M	2	N	No	No	N	23.4	0.9	N	170	100	N	N	80	24	1.2	148	4	N	N
59	54/F	4	N	No	No	N	25.6	0.98	I	170	110	N	N	104	28	0.8	133	4.8	N	N
60	56/F	3	N	No	No	N	24.6	0.92	N	180	100	N	N	112	31	1.2	134	3.6	N	N
61	60/F	2	N	No	No	N	21.3	0.9	N	180	110	N	N	92	28	0.8	132	5	N	N
62	60/F	2	N	No	No	N	23.2	0.85	N	170	100	N	N	112	32	1.1	138	3.5	N	N
63	59/M	1	N	Y	No	N	22.3	0.85	I	180	100	N	N	108	36	1.1	141	4.6	N	N
64	58/F	2	N	No	No	N	21	0.91	N	170	100	N	N	108	42	0.9	139	4.6	L	N
65	52/M	2	N	Y	No	N	23.8	0.91	I	200	110	N	N	120	43	1.2	146	3.9	L	N
66	48/M	3	N	Y	Y	H	20.7	0.93	I	170	100	N	N	92	36	1.1	153	3.4	N	N
67	60/M	2	N	No	No	N	22.1	0.93	N	150	100	N	N	86	18	0.9	144	5	N	N
68	54/F	2	N	No	No	N	25.5	1	N	170	100	N	N	72	20	1	138	4	N	N

Sl. No	Age / Sex	Symptoms	P / H	Smoking	Alcohol	F / H	BMI	WHR	Fundus	SBP	DBP	SE	UR	Bl. Glucose	Bl. Urea	Sr. Creatinine	Sr. Sodium	Sr. Potassium	ECG	CXR
69	59/M	2	N	Y	No	N	18.2	0.88	N	170	100	N	N	78	20	0.7	149	3.9	N	N
70	52/M	4	N	Y	No	N	25.7	0.95	N	200	110	N	N	92	26	1	153	3.8	L	C
71	51/F	0	N	No	No	N	19.1	0.79	N	120	80	N	N	73	20	0.8	136	4	N	N
72	43/M	0	N	Y	No	N	20	0.81	N	110	70	N	N	80	21	0.7	137	4.1	N	N
73	45/M	0	N	Y	Y	N	18.9	0.8	N	100	70	N	N	82	19	0.7	146	4.7	N	N
74	48/M	0	N	No	Y	N	17.7	0.87	N	90	70	N	N	90	18	0.8	140	3.9	N	N
75	42/M	0	N	Y	Y	N	17.3	0.86	N	100	70	N	N	70	20	0.7	144	4.6	N	N
76	46/M	0	N	No	No	N	22.6	0.97	N	110	80	N	N	80	22	0.8	140	4.8	N	N
77	54/M	0	N	No	Y	D	19.3	0.91	N	110	80	N	N	84	24	0.8	138	4.6	N	N
78	49/M	0	N	Y	No	N	20.2	0.91	N	110	70	N	N	70	20	0.7	144	4.1	N	N
79	50/M	0	N	No	No	N	23.2	0.88	N	110	70	N	N	74	21	0.8	139	4.2	N	N
80	45/M	0	N	No	No	N	21.3	0.8	N	110	70	N	N	84	22	0.7	134	4.9	N	N
81	46/F	0	N	No	No	N	26.4	0.8	N	110	70	N	N	87	16	0.6	140	4.2	N	N
82	56/M	0	N	Y	Y	N	20.5	0.96	N	100	70	N	N	87	22	0.7	139	4.1	N	N
83	45/F	0	N	No	No	N	20.3	0.82	N	100	70	N	N	99	20	0.8	138	4.8	N	N
84	60/M	0	N	Y	Y	N	19.8	0.83	N	110	70	N	N	69	20	0.8	138	4.5	N	N
85	55/M	0	N	Y	Y	N	17.9	0.9	N	100	70	N	N	80	36	1.2	138	4.4	N	N

CXR	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
ECG	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Sr. Potassium	4.6	4.7	4.1	4.2	3.8	4.2	4	4.1	4.2	3.9	4.1	4.2	3.8	4.8	4
Sr. Sodium	136	137	146	145	144	140	138	144	139	144	140	139	138	138	138
Sr. Creatinine	1.1	1	1.1	0.8	0.6	0.9	0.8	1.1	0.8	1.1	1.1	1	0.8	1.1	1.1
Bl. Urea	28	32	39	28	30	36	24	28	21	32	36	42	21	42	36
Bl. Glucose	113	108	118	108	96	84	84	114	104	120	102	98	109	112	96
UR	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
SE	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
DBP	78	60	70	70	74	80	70	70	70	70	70	70	70	70	70
SBP	116	100	100	110	110	110	110	110	110	100	110	100	110	100	110
Fundus	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
WHR	0.84	0.9	0.88	0.87	0.86	0.97	0.97	0.98	0.95	0.8	0.98	0.96	0.93	0.94	0.9
BMI	20.5	23.7	23	22.7	21.2	22.6	22.3	22.9	24.4	23.2	21.2	21.9	23.8	22	20.8
F / H	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Alcohol	Y	No	Y	No	No	No	No	No	No	No	Y	No	No	No	No
Smoking	Y	Y	Y	No	No	No	No	Y	No	No	Y	Y	No	Y	No
P / H	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Symptoms	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Age / Sex	51/M	43/M	45/M	48/M	52/F	53/F	54/F	62/M	50/F	45/F	46/M	45/M	48/F	60/M	54/F
Sl. No	86	87	88	89	90	91	92	93	94	95	96	97	98	99	100

0 – no symptom 1 – headache 2 – giddiness 3 – chest pain 4 – palpitation 5 – dyspnoea
Y – Yes N – normal M – male F- female I – grade I ht retinopathy L – LVH
B – LAHB C – cardiomegaly F/H – family history P/H – past history